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SURGERY

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SURGERY

VOL. 27 JUNE, 1950 No. 6

Original Communications

THE RELATION OF ACID SECRETION TO PEPTIC ULCERATION AND ITS TREATMENT

SIR HENEAGE OGILVIE, LONDON, ENGLAND

(From Guy's Hospital)

THE exact location of the seat of the soul has been argued among doctors, philosophers, and divines throughout the ages. The stomach has a greater claim than most, being traditionally associated with elation and depression. "Whoso hath a proud look and a high stomach, I will not suffer him," says the psalmist. "He which hath no stomach to this fight, let him depart," says Henry V. Despondency, on the other hand, is described as a "sinking in the pit of the stomach." And these phrases are not mere rhetoric. Barclay, during one of his earliest studies on the position of the viscera in man, reported that when a door slammed behind a patient he was observing on the screen he saw the greater curvature of the stomach suddenly drop to the pelvis. Bennett and Ryle found that the suggestion of depression to a normal student during a test meal led to a profound drop in the acid level, and to delay in emptying. The intricate relation between the behavior of the stomach and the emotions has been more recently studied by Wolf and Wolff, whose subject, Tom, made up for his very indifferent talents as a laboratory attendant by the double qualification of a gastric fistula and a suggestible temperament. Rage and frustration made Tom's gastric mucosa blush and pour out more juices with a high acid level; fear, disappointment, and depression made it go pale and secrete small amounts with little acid.

While the stomach is under the influence of the emotions, the one emotion (for it is more than a sensation) that would be considered to be purely gastric, that of appetite, seems to have nothing to do with the stomach at all. A man with cancer of the stomach loses his appetite, but if he undergoes total gastrectomy with anastomosis of the jejunum to the esophagus he regains it, longs for his meals, and eats every kind of food with relish. The sensation of appetite is thus somatic or metabolic in origin, whereas its opposites, nausea or distaste for food, arise in the stomach.

The man in the street knows that the stomach contains acid, but he feels that nature has somehow slipped up in filling his insides with the same chemical

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that he puts in the batteries of his car, that etchers use to eat away copper, and that murderers use to dispose of the bodies of their victims. He is ready to explain any digestive upset as "acidity" and to treat it with any nostrum that promises to neutralize the acid. The men who sell stomach powders have an inexhaustible market, and they prosper exceedingly.

Acid in the stomach is normal. The average healthy young man or woman, as typified by the average medical student who provides the human guinea pig for most clinical physiologic research, has a high acid level, and the fitter and the happier he is, the higher the level seems to be; only when he is depressed, ill, or has undergone an operation does the level fall. Yet in a certain number of exactly comparable young people, coming from the same class and equally healthy, complete achlorhydria is found. Neither a high acid nor a low acid is necessarily the cause of disease or even of ill health, but departures from the average are associated with certain pathologic processes, as hyperchlorhydria with peptic ulceration, and achlorhydria with gastritis, diarrhea, pernicious anemia, and cancer of the stomach.

Peptic ulcers are found only in structures exposed to the action of gastric juice containing hydrochloric acid. They are usually limited to the part of the alimentary tract in which unneutralized hydrochloric acid is normally found, that is the stomach and first part of the duodenum in anatomic parlance, the stretch from the cardiac opening to the bile papilla speaking physiologically, the foregut in the embryologic sense. They may occur in other parts of the intestine where hydrochloric acid is secreted by areas of ectopic gastric mucosa, such as Meckel's diverticulum, or where gastric juice trespasses its normal gates, as in the esophagus when the cardia is ineffective, as it often is in the condition of short esophagus or hiatus hernia. Peptic ulcers frequently affect any organ that is exposed by surgical anastomosis to gastric juice; the jejunum after gastrojejunostomy and occasionally after gastrectomy, the esophagus after cardioplasty or esophagogastrostomy, the gall bladder after cholecystogastrostomy, and the skin tube after antethoracic esophagoplasty. They do not occur when hydrochloric acid secretion is absent or lost through disease.

It is thus easy to assume, as indeed it is assumed in most textbooks, that acid is the cause of ulcer. It is as logical to assume that the heartbeat is the cause of hypertension, that secretion of urine is the cause of stone, that thought is the cause of lunacy. The secretion of hydrochloric acid by the gastric mucosa is a normal process. The digestion of meat fibers by pepsin depends on the presence of free hydrochloric acid, and ceases in achlorhydria. The germicidal action of normal gastric juice is due to its hydrochloric acid content. All healthy dogs and all healthy men have a high acid level in their gastric juice, yet no dogs and few men develop ulcers. The immunity of the normal foregut from autodigestion by the powerful ferment and the strong mineral acid it secretes is due to two things; first, that gastric juice is poured out only when a meal is being eaten or about to be eaten, so that the acid is buffered by food, and second, that the surface of the gastric mucous membrane is composed of a layer of nonsecreting epithelial cells interspersed with mucous cells, and is normally covered by a layer of mucus. We must therefore look for the cause

of ulcer, not in the fact that the gastric juice contains acid, but in some increase in the digestive power of the juice, some disturbance in the normal relation of secretion to meals, or some failure in the protective mechanism.

Acute ulcers may be much more common than we know, because they are unrecognizable by radiography, and they are seen only when the stomach is inspected by the gastroscope, at operation, or at post-mortem—that is, when they cause symptoms sufficiently severe to justify one of these interventions. They may be the pathologic basis of many an unexplained bellyache. Acute ulcers are multiple, and are found in any part of the stomach. They are usually associated with an acute infection, some toxic condition, or a nutritional deficiency. They are at first breaches of the mucous membrane only, roughly circular with sharp-cut edges, and they tend to erode the large vessels that run in the submucous coat and give rise to severe hemorrhage. If they persist, they involve the other coats in turn, but in the stomach, at any rate, they rarely perforate. Under the microscope they show edema and cell destruction, but few of the characteristic changes of an inflammatory process. The majority heal without leaving a trace. Most duodenal ulcers that perforate are of this same acute edematous noninflammatory type, but they are not found often in association with acute gastric ulcers.

Chronic peptic ulcers are observed only in certain sites that have come to be named the ulcer-bearing areas. In the stomach they occur on the lesser curve, extending as they enlarge onto the posterior wall and to a less extent onto the adjacent anterior wall, but they are rare at the cardiac end of the lesser curve, and so rare in the prepyloric inch and on the pylorus itself that ulcers here must be regarded as probably malignant if they remain unhealed for more than a few weeks. In the duodenum they usually start on the anterior wall about one-quarter to one-half inch beyond the pylorus, but with time they tend to enlarge, to become multiple, and to extend downward, even as far as the bile papilla. Chronic gastric ulcers, at any rate in those countries where statistics are kept, are very much less common than duodenal ulcers, and their incidence in the two sexes is about equal. Chronic duodenal ulcers are about four times as common in men as in women and they show a remarkable geographical distribution which has not as yet been clearly linked with any possible etiological factor.

I will not attempt to recapitulate the experimental work on ulcer production. Two years ago I read as assessor a thesis for the degree of Master of Surgery which analyzed nearly 200 papers on this subject. I laid it down with the impression that most of the writers of these papers had produced ulcers in animals only by forms of mechanical, thermal, chemical, toxic, or bacterial assault so massive that they had no relation to any causes that were possibly operative in man, and with the conviction that these causes must still be sought by clinical observation and experiment. Some of the theories emanating from the laboratory hardly merit consideration. Rosenow's interesting work on the elective action of streptococci does not seem to have been accepted as bearing in any direct way on the ulcer problem in human beings. "Thrombosis of end arteries" is a pathologic cliche, like "that blessed word Mesopotamia." There is

a complete, or nearly complete, watershed at the pylorus between the arteries of the stomach and duodenum, but the pylorus is the one place in the acid territory in which ulcers never occur. There are no end arteries in the stomach or duodenum; on the contrary a life's experience of gastric surgery has taught me that the divided stomach will bleed freely many inches beyond the point where its vessels have been tied, and that the duodenum can be cleared beyond the plane of proposed section for a length equal to its breadth without any danger that the cut edge will slough. Where end arteries occur in the body they are in organs constructed on the system of units and they do not thrombose in people as healthy as most ulcer patients. Then there is the time-honored theory that chronic ulcers are found on the lesser curvature because it is the food channel or magenstrasse and therefore subjected to trauma. Does anyone believe this? The lesser curve is a short cut for fluids, but solids are churned about in the stomach until they are no longer solids. If we are kept awake at night by the fear that fish bones are rushing down our magenstrasse tearing off bits of mucous membrane as they go, we can derive comfort from the knowledge that the only place they are likely to scrape hard enough to cause trauma is the pylorus and prepyloric inch, a place where ulcers are almost unknown.

From clinical experience we can agree upon some pointers on etiology that must color our evaluation of experimental studies. There is a nervous element, using the word in its vague popular sense, in the causation of duodenal ulcer, at any rate among Europeans. The intelligent, restless, frustrated outlook typical of the duodenal diathesis is undoubted, but it is not clear whether or not the mechanism by which it produces the lesion is related in any way to that by which stimulation of centers in the midbrain produces ulcers in animals. Local trauma to the stomach wall clearly contributes to the production of those gastric ulcers that form at the site of constriction in diaphragmatic hernia, but this form of trauma has no counterpart in the ordinary ulcer patient. Toxins again, those of certain drugs applied to the surface and those of uremia acting through the blood stream, can cause acute gastric ulcers in human beings, but such insults are too unusual to have any important bearing on the etiology of ulcers in the apparently healthy. An acute infection, such as a burn or suppurative appendicitis, may be followed by an ulcer, but nearly always an acute single duodenal one that perforates or bleeds, seldom a chronic one. Malnutrition is probably a contributory factor to the development of gastric ulcers, which in Europe appear to be more common in the underprivileged classes. In the ulcer belt of Southern India, where the proportion of patients suffering from peptic ulceration in relation to the total surgical admissions to hospital is ten times as high as that in other parts of India, malnutrition has been cited as the cause. The peasantry in the ulcer belt are too poor to worry and to smoke, and too placid to suffer from frustration, yet they develop massive duodenal ulcers beside which those of the frustrated London stockbroker are small lesions. Their diet consists almost wholly of rice in some districts, of tapioca in others, and their protein intake averages may be as low as 100 to 150 Gm. a week. Lest protein deficiency should be accepted without question as the cause of these ulcers, it should be remembered that in many other non-

ulcerous districts of India protein shortage is no less severe, and that the peasants of Egypt and the tribesmen of Africa, who suffer from a similar deficiency, do not develop peptic ulcers. It may be that the South Indian ulcers are caused, not by a deficiency, but by the pepper which is added to make this revolting fare less unpalatable. Of all observations that rest securely on clinical experience as well as experiment, the most constant is that gastric juice containing free hydrochloric acid, when acting on any surface other than intact gastric mucosa, will produce on ulcer.

Do acute ulcers go on to become chronic ulcers? There is every reason why they should, for, whatever their cause of origin, they provide a site at which the normal protective mechanism has been breached and the gastric juice can gain access to the unprotected deeper layers. Yet the mere fact that acute ulcers are multiple and found all over the stomach, while chronic ulcers are single and found only on the lesser curve, proves that, at any rate, the majority of acute ulcers do not become chronic. Do chronic ulcers start as acute ulcers, and if they do why should ulcers persist and become chronic only in certain situations? Here it is easier to suggest an explanation than to prove its correctness. The functional characteristic of the ulcer-bearing areas, the lesser curve of the stomach and the first inch of the duodenum, is that they are areas of no movement. The lesser curve varies little in site, length, and curve in fasting and in repletion, whereas the rest of the stomach expands readily to the size needed to accommodate the meal. The first inch of the duodenum is a reservoir of a transient sort, holding the emerging chyme long enough to show on an x-ray film as a cap, whereas the remaining eleven inches pass the food along in rapid peristaltic waves. Any surgeon interested in gastric work will agree that the mucous membrane over these still areas is bound to the muscle coats, while in the mobile areas it is loose, folded, and freely movable on an areolar submucous layer. When the stomach is opened the mucous covering of the lesser curve is seen to be smooth and fixed, while that over the anterior and posterior walls is as convoluted as the cerebral cortex, and pouts out from the cut in loose folds that are a nuisance in forming an anastomosis. The mucous lining of the duodenum, where we cut it to do a Billroth I anastomosis, is as smooth as an inner tube; two inches lower where we expose it to look for the bile papilla it is as loose and folded as the socks of a professor.

Here then is the possible explanation of why acute ulcers should heal in the body of the stomach, yet become chronic lesions in the duodenum and over the lesser curve. In each case a punched hole is made in the protective covering exposing the deeper layers to digestion; but where the mucous membrane is loose the edges of the hole fall together as the stomach empties and it heals as rapidly as would a small burn on the eyelid; where the mucous membrane is fixed the hole remains open as would a small burn on the bridge of the nose, and digestion gets to work before it can heal.

If we were able to inspect the lining of our whole ailmentary canal with the constant scrutiny and minute attention that a girl devotes to studying her complexion, we should probably see that our inner surface, like our outer one, was constantly showing small blemishes due to abrasions, infections, dietetic

indiscretions, drug rashes, exanthemas, or allergies, and that the majority of these healed rapidly without leaving a trace. But when they happened to occur on a particular site, the fixed areas of the stomach and duodenum, when they involved a breach of the surface or damage sufficient to inhibit the protective mechanism, they were liable to become chronic ulcers if our acid secretion was normal or high. According to this view, the relation of hydrochloric acid to peptic ulceration is that it is not the cause of origin, but the cause of persistence of lesions whose first cause is a local damage to the mucous membrane from any of a number of different harmful agents. Nevertheless, the surgical treatment of ulcer demands two things, the removal of the ulcer or its removal from the pathway of gastric juice, and the reduction of gastric acidity to a level that will prevent future ulceration.

Gastric juice contains hydrochloric acid, pepsin, rennin, lipase, the intrinsic factor, and mucin. It is secreted by the gastric mucous membrane, in which three types of glands are recognized: the cardiac, the principal, and the pyloric. The cardiac and pyloric glands are similar in appearance and probably in function; they produce an alkaline secretion rich in mucus, and the pyloric glands, at any rate, secrete the intrinsic factor. The principal glands secrete pepsin and hydrochloric acid, the acid being produced by the wedge-shaped parietal cells that lie against the basement membrane of the crypts. In man the cardiac glands occupy only a small strip around the cardiac orifice, and the pyloric glands occupy a territory about two inches long, proximal to the pylorus. The principal glands occupy the rest of the stomach, but the mucous membrane containing them is thicker and more complex, and therefore probably more productive in the body than in the fundus.

Very little is known about the composition of gastric juice in the lower animals. The stomach first makes its appearance in fishes, and in them pepsin and hydrochloric acid are first encountered. In fishes the hydrochloric acid serves the threefold purpose of killing their prey, killing bacteria that have been swallowed, and dissolving the calcareous exoskeletons of swallowed crustaceans and other forms of marine life. Gastric secretion in fishes and amphibians is not under the control of the nervous system, and it is probably called forth by the mechanical and chemical stimulus of swallowed food. In birds there is evidence of a nervous control of gastric secretion; thus a duck, fed habitually at the sound of a bell, will secrete gastric juice when the bell is rung even though no food is given, much after the manner of Pavlov's dogs. Most of the teaching on gastric secretion in man is based on experiments on the dog, an animal with similar but not identical social and dietetic habits. We might learn much if we knew how the gastric juice of the chicken, that eats corn, differs from that of the cormorant, that eats fish, and in what medium the koala bear digests a diet consisting of nothing but eucalyptus leaves.

From the dog we learn that there are two phases in gastric secretion, the nervous and the hormonal. The nervous or psychic phase is induced by the sight, smell, or thought of food. It is abolished by section of the vagi. In the dog this phase starts from five to ten minutes after the appropriate stimulus, and reaches a peak in one hour. The second phase has two components, a

gastric and an intestinal, which depend on the presence of food in the stomach or intestine. A hormone, gastrin, liberated in the pyloric mucosa by the presence of food and absorbed into the blood stream, stimulates a flow of gastric juice that starts after a latent interval of about one-half hour and reaches its peak in about two hours. A similar hormone is evoked when food enters the small bowel. The second or hormonal phase of gastric secretion is not inhibited by denervation of the stomach. A hormone that inhibits gastric secretion, enterogastrone, is produced when fat is introduced into the duodenum; its action is not affected by denervation of the stomach.

I have already suggested that we must seek the cause of peptic ulceration not in the fact that the stomach contains acid, but in some disturbance of the normal mechanism of secretion and protection—in some increase in the digestive power of the juice, some alteration in the time relation between secretion and meals, or some failure in the mechanism of protection. So far I have referred only to the last aspect, that is, to agents that may cause local morbidity and wreck local defense.

A high level of acid is common in ulcer patients. Aird says that 90 per cent of patients suffering from duodenal ulcer have hyperchlorhydria. Hurst emphasized the tendency for hyperchlorhydria and ulcers to run in families. Cinchophen, the injection of histamine in beeswax (Code and Varco, 1940), electrical stimulation of the vagi in the thorax (Stahnke, 1924), and the continuous injection of acetylcholine (Meckels, 1937), all of which stimulate the secretion of gastric juice, have been able to produce chronic peptic ulcers in animals. Yet a high acid level in man, provided the gastric juice is secreted only in response to meals, is hardly likely to originate an ulcer. It is only more potent than a low acid juice to convert a lesion arising otherwise into an ulcer. Even so a boy of 16 years, piloting a motorcycle at ninety miles an hour along a road, is no more likely to meet a cow than a timid dame of 60 years, chugging along at sixteen miles an hour in a model T Ford, but if he does he is more likely to get hurt.

The secretion of gastric juice into a stomach unprotected by food is a very probable cause of primary ulceration, and an undoubted cause of postoperative ulceration. Many of the experiments I have referred to, such as injection of histamine in beeswax and electrical stimulation of the vagi, cause ulcers because they lead to the secretion of acid into a stomach unprotected by food rather than because the acid is higher than normal. Mann and Hohn (1932) produced chronic lesser curve ulcers by instilling 0.14 per cent hydrochloric acid drop by drop for eight hours a day through a gastric fistula. Constant acid secretion is a more significant abnormality in patients with peptic ulcers than high acid secretion. A large volume of resting juice is characteristic of fractional test meals in ulcer patients. Dragstedt, in 1947, investigated the night secretion in normal subjects and in patients with duodenal ulcer by applying continuous suction to a tube left in the stomach overnight. He found that the control individuals secreted from 250 to 400 c.c. of juice with from 0 to 20 units of free acid, whereas the duodenal patients secreted from 600 to 2,500 c.c. of

juice with from 40 to 110 units of free acid. The significance of this experiment lies in the difference between the two groups rather than in the figures, for the indwelling tube probably acted as an irritant in both groups.

Possibly the strongest argument in favor of the view that unbuffered acid, rather than acid in itself, is the cause of peptic ulceration, may be found in the disastrous results of exclusion gastrectomy. I was the first to point out the dangers of this operation which I had previously advocated, and I can at any rate claim the distinction that, having reasoned wrongly and given foolish advice, I publicly recanted and warned others not to be led astray by my folly. In some 400 of my gastrectomies for ulcer in which the pyloric antrum was removed, none, as far as I know, have had recurrent ulcers, but in a series of twenty-two done about fifteen years ago by an identical method except that the pyloric end of the stomach was not removed, six had jejunal ulcers proved at operation and three more had pain that was relieved by a course of medical treatment. The probable explanation of these ulcers is that after exclusion gastrectomy the hormonal phase of gastric secretion comes into operation after all food has left, and for one hour or more acid juice is poured abundantly onto an empty stomach and over an unprotected stoma.

Peptic ulceration is a disabling rather than a mortal disease, and surgery is not the only method of treatment. Any operation that is propounded for its cure must therefore be safe in its performance, and satisfactory and lasting in its results. Safety depends entirely upon the skill, experience, and devotion of the team that cares for the patient from the time when he presents himself as a suffering supplicant to the time when he is discharged as a satisfied customer; more deaths are caused in gastric surgery by inadequate preparation, poor anesthesia, and slipshod aftercare than by unskillful technique. The quality of the results depends upon the design and the execution of the operation. An operation designed to cure the symptoms of a peptic ulcer must at any rate satisfy three criteria: it must heal or remove the ulcer, it must overcome the effects of the ulcer, and it must prevent recurrence. In practice this last requirement involves the permanent abolition or reduction of gastric acidity.

I do not propose to discuss the medical treatment of peptic ulcer. I will say only that most surgeons believe that the sedative drugs, particularly phenobarbitone, are of greater value than the alkalies, and that rest in bed is more important than diet. Medical treatment is satisfactory only when it leads to healing within one year, or at any rate before permanent scarring has taken place. On the whole the results are poor. Krarup analyzed 1,100 patients treated medically over a period of five years. The results were: recovered, 20 per cent; recovered with one relapse, 3 per cent; improved but not symptom-free, 29 per cent; unsatisfactory, 28 per cent; died, 15 per cent; not traced, 6 per cent. St. John and Flood (1939) found that in a group of 225 patients treated medically, 65 per cent relapsed within two years.

Of surgical procedures, excision of the ulcer without acid reduction must be condemned. It is useless. It is still done in common with many other useless operations, for a bad surgeon is born every minute and bad surgery is easy and profitable, but it is still useless.

Reduction of the free hydrochloric acid in the ulcer-bearing areas to a safe level has been attempted by surgical means in four main ways:

1. By neutralization, leaving the mechanism of acid production intact; the various short-circuiting operations make this attempt

2. By reducing drastically the blood supply to the stomach, thus attacking the glandular mechanism of secretion, and leaving the control mechanism unaffected

3. By removing the stimuli to gastric secretion
   (a) the nervous stimulus alone, by vagotomy
   (b) both nervous and hormonal stimuli by vagotomy combined with pylorectomy

4. By an attack on the control and glandular mechanisms together in partial gastrectomy

The short-circuiting operations, gastrojejunostomy, gastroduodenostomy, and pyloroplasty are wrong in theory and unsuccessful in practice. As an individual I like them, for I make a good living by resecting other surgeon's jejunal ulcers and gastrocolic fistulas, but as a gastroenterologist I must condemn them.

The theory underlying these operations is that neutralization by the entry of alkaline duodenal contents into the stomach at the end of a meal is a normal process, and that a failure in this reflux is a cause of ulceration. That one organ should call on another for help in doing its job would be unusual in the body. In the alimentary system the salivary glands, the pancreas, the intestinal glands, and the large intestine all do their allotted task and hand on the result to the next department. It is more than likely that the stomach, which, unlike the other departments, locks the doors at each end while it is busy, should do the same. Observation tells us that bile may be found in the stomach during test meals, but inconstantly and in small amounts. Avery Jones told me that during gastroscopy he has seen bubbles come back through the pylorus, but not bile. During radioscopy, regurgitation of barium is seen only in duodenal ileus. The effect of short-circuiting operations in reducing the acid level is merely that of any handling that interferes with function. After one year the acid level is as high as before, often higher (Heuer, 1944). These operations overcome stasis when it is present, but since they do nothing to hinder acid production, but indeed encourage it by relieving the gastritis that stasis has caused, they allow the hormonal phase of acid secretion to pour into an empty stomach. In practice gastrojejunostomy is followed by unsatisfactory results in 40 per cent of cases, among which are 10 per cent of gastrojejunal ulcers. Lewisohn (1925) reported 80 per cent of failures and 34 per cent of anastomotic ulcers, and pathologists give a similar figure based on post-mortem investigations. An incidence of recurrent ulceration in 10 per cent of cases is accepted by most gastric surgeons who are not selling their own figures.

Vasoligation may, I think, be dismissed as an unfortunate experiment that is now dead. The mere fact that no one in Manchester has followed Wilson Hey and no one in India has followed Somervell is, in my view, sufficient condemnation.

Division of the vagi for various indications has been tried for many years, but vagotomy as a reasoned procedure, based on the results of animal experiments, was introduced for the treatment of peptic ulceration by Dragstedt in 1945. It is unfortunate that the greed of the popular scientific magazines for novelty thrust this operation into the limelight before it could be appraised calmly. Vagotomy abolishes the psychic secretion of gastric juice and the acid response to hypoglycemia. It abolishes the pain of ulcer and leads to healing, temporarily at any rate, in the majority.

The criticisms of vagotomy are two. First, it does nothing to overcome the scarring, fixation, and stenosis due to the ulcer, but on the contrary, by diminishing gastric motility, it tends to aggravate the stasis that may have been caused. For this reason it has been found necessary in many instances to combine vagotomy with some form of short circuit or resection. Second, it does not abolish the hormonal phase of gastric secretion, and when a short circuit has also been done, the stomach is empty when this phase comes into action. Saltzstein (1947) has found that vagotomy fails to prevent the development of jejunal ulceration in dogs after the Mann-Williamson operation. Lillehei and Lewis find that much of the protection of vagotomy is lost if a drainage operation is done at the same time.

Because of these criticisms, some surgeons are combining vagotomy with pylorectomy or hemigastrectomy, thus abolishing both phases of gastric secretion and producing complete and permanent achylia. Here is an operation that cannot be judged until it has been given extended trial. It is clearly a simpler and therefore a safer operation than a high gastrectomy.

Last, we must consider radical gastrectomy, the operation which I believe still to be the best means of removing the ulcer and reducing the acid to a safe level, if it is properly performed. I do not wish to obtrude my personal fads, which are many. I detest the Polya operation, because it often leads to the dumping syndrome or to proximal loop distention. I abominate all anterior anastomoses except for cancer, because they distort the proximal loop and make the stomach dance attendance on the vagaries of the colon, instead of the other way around as it should be. I will merely state what I believe to be the five-point program essential to success.

1. The whole pyloric mucous membrane must be removed to abolish hormonal secretion.

2. The perigastric tissues, nerves, and vessels must be divided on the lesser curve at or near the level of the cardia to reduce nervous secretion to about 20 per cent of its former level, and to leave the reduced stomach hanging free.

3. The body of the stomach containing the thick acid-secreting mucosa must be resected.

4. The stoma must be no larger than the diameter of the duodenum or jejunum to which it is anastomosed.

5. Food after leaving the stomach must be able to pass onward only.

I would suggest that we do not wish to abolish gastric digestion entirely. Vagotomy with pylorectomy leaves a moderate-sized stomach but produces achlorhydria, and we should ask ourselves if that is a wise thing. Patients with achlorhydria are often symptomless, but they have lost one phase of normal digestion, they have lost the bacterial protection of gastric juice, they are more prone than others to alimentary upsets and attacks of diarrhea, and they are probably more prone than the average population to cancer of the stomach. Kaplan and Rigler (1945) reported 18 cases of cancer of the stomach (7 per cent) and 17 cases of simple tumors, which in the stomach are often precancerous (6.5 per cent), or 13.5 per cent of tumors in all, in a series of 259 patients with pernicious anemia. Bourne (1948) found 3 cancers and 2 nodules of doubtful nature in a smaller series of 15 patients. There were 431,729 people over the age of 30 who died in Britain in 1946, of whom 13,777 died of cancer of the stomach, so that the incidence in the population as a whole is about 3.2 per cent. The very high gastrectomy advocated by Visick, whose recent death was a tragedy to British surgery, was said by him to produce achlorhydria in 98 per cent of patients. Such a high gastrectomy, like a radical Polya operation, also removes the stomach as a reservoir, converting it into a funnel that hands on the food straight from esophagus to jejunum.

I believe that a gastrectomy, planned on the five criteria enumerated here, should leave a stomach with a capacity of nearly a pint, secreting acid of no more than 20 to 30 per cent N/10 NaOH concentration, secreting it only in response to a meal, and holding the meal for about one-half hour. I believe that such an operation should carry a mortality of less than 2 per cent. I believe also that it should give a complete cure in at least 90 per cent with no jejunal ulcers. The not quite perfect results are seen in those patients who complain of hurry afterward, usually nervous individuals with small lesions, who were operated upon because the physicians were sick of them. Such individuals with such lesions provide, in my opinion, the only subjects in whom vagotomy may be the right treatment.

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PRELIMINARY REPORT ON A NEW METHOD OF INTESTINAL INTUBATION WITH THE AID OF A FLEXIBLE STYLET WITH CONTROLLABLE TIP

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THE intestinal intubation therapy for obstruction of the bowel is efficacious clinically, but the successful passage of the tube is difficult, time consuming, and frequently unsuccessful. To obviate these obstacles, a technique of rapid intubation employing a flexible stylet with a controllable tip has been developed at this clinic.

This method of intubation facilitates both preoperative preparation of the obstructed patient and early nonoperative treatment.[1] The stylet method of intubation has also been of value in the treatment of postoperative distention, for the presence of bowel motility is not important in the placement of a tube in the small bowel by this method.

In many cases we have been able to place the end of the tube beyond the ligament of Treitz (Fig. 1).

THE STYLET

The controllable tip (1¼ inches long) is made of a flat spring enclosed within a coiled spring. There are two fine wires attached to the point of the controllable tip which pass on either side of the flat spring, inside the coiled spring, and on into the inner coil of the stylet.

The inner coil (5 inches long) is a coiled flat wire. This is of light construction and forms a flexible column through which the fine wires pass from the controllable tip to the body.

The body is a more heavily constructed coil spring (4½ feet long). Through it pass the two fine wires from the tip, and a third wire which is attached to the junction of the body and the inner coil.

The handle is attached to the body. The wires from the controllable tip traverse the handle and are attached to the large lever. Moving this lever through an angle of 60 degrees from the mid-position places tension on one wire and turns the controllable tip through an arc of 90 degrees to the same side. Moving the lever arm to the opposite side points the controllable tip in the opposite direction; thus the controllable tip may be moved through an arc of 180 degrees.

A thumb lever on the side of the handle is attached to the third wire. Pressure on this lever tightens the wire and stiffens the body of the stylet.

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THE TUBE

The tube, devised by one of us (G. A. S.), is 9 feet long and is made of Koroseal. A small Lucite plug is cemented into its end. Two holes, one on each side of the plug, connect with the lumen of the tube. A latex balloon is tied onto the tubing just behind the Lucite plug.[4] A small plastic tube is attached to the balloon to inflate it with air. A hole is cut in the wall of the tube 4 feet, 9 inches from the balloon to admit the stylet. This hole is later plugged by a small metal tubing insert. The stylet is easily inserted and extracted from this tube.

THE TECHNIQUE OF INTUBATION

The tube and stylet are lubricated with instrument oil. The stylet is introduced through the hole in the side of the tube to 1 inch from the Lucite plug. The patient is positioned supine on the fluoroscopy table with a Wangensteen duodenal tube[2] in the stomach. The stomach is distended with 800 to 1,000 c.c.

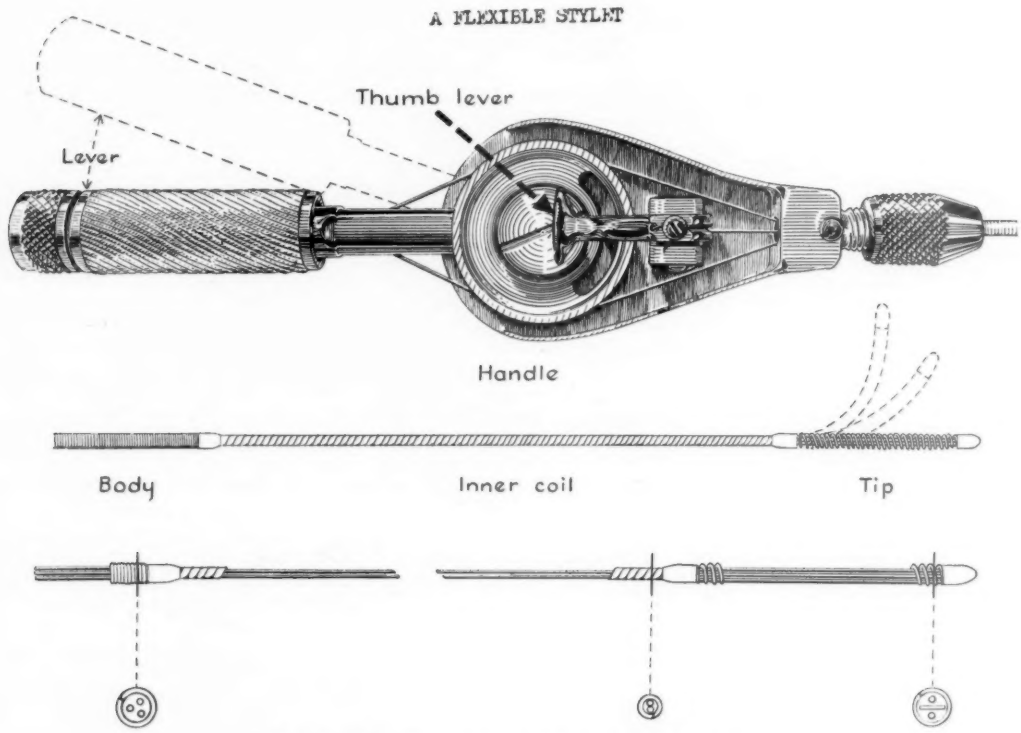

Fig. 1.—Flexible stylet with controllable tip.

of air.[3] The tube, with the stylet in place, is introduced through the mouth or nose into the cardiac portion of the stomach. The controllable tip is turned to point to the patient's right, and the tube and stylet are advanced along the greater curvature of the stomach until the end of the tube lies at the pylorus. The controllable tip is then manipulated until there is no curve present. Pressure is made on the thumb lever to stiffen the body of the stylet, and the tube is advanced into the duodenum. The stylet is withdrawn from the tube; the balloon is inflated with 20 c.c. of air, and the hole in the side of the tube through

which the stylet was inserted is closed with the metal tubing insert. All manipulations are done under fluoroscopy; however, the fluoroscope need be on only for brief periods during the procedure (Fig. 2).

RESULTS

Twenty-two of twenty-three patients with moderate to severe intestinal obstruction have been intubated. The patient in whom intubation failed had adhesions about the duodenum from a previous laparotomy. She was treated by lysis of the adhesions and the tube was then manipulated into the small bowel (Table I).

TABLE I. CASES OF INTESTINAL OBSTRUCTION INTUBATED WITH STYLET AND TUBE

INTUBATION TIME (IN MINUTES)	PORTION REACHED BY STYLET INTUBATION	PREVIOUS METHODS OF INTUBATION	DIAGNOSIS
4	Jejunum	Successful Wild tube passage in 2 hours	Generalized carcinomatosis
10	Jejunum	Successful Wild tube passage in 2 hours	Generalized carcinomatosis
20	3rd portion duodenum		Cancer of terminal ileum
30			Cancer of pelvis
3	Jejunum		Postoperative ileus
5	Jejunum	15 hour Wild tube failure	Obstruction with previous gastric resection
5	Jejunum	15 hour Wild tube failure	Obstruction with previous gastric resection
15	3rd portion duodenum	7 hour Wild tube failure	Fecal fistulas, RLQ
20	Failure	20 hour Wild tube failure	Postoperative adhesions about duodenum
7	3rd portion duodenum		Recurrent obstruction
*15	Ligament of Treitz	Stylet and tube passed in 10 minutes	Cancer of pancreas with metastases
10	3rd portion duodenum	15 hour Miller-Abbott tube failure	Cancer of large bowel
5	3rd portion duodenum		Postoperative ileus
10	3rd portion duodenum	Several days and methods failed	Recurrent obstruction
10	3rd portion duodenum		Chronic obstruction
5	3rd portion duodenum		Transverse myelitis
*5	Ligament of Treitz		Strangulated ileum
5	Ligament of Treitz		Perforated ileum with peritonitis
20	Ligament of Treitz	24 hour Wild tube failure	70 day postoperative ileus
10	2nd portion duodenum		8 day postoperative ileus
10	18 inches past ligament of Treitz		Chronic partial obstruction
15	6 inches past ligament of Treitz	16 hour Miller-Abbott tube failure	Acute obstruction upper jejunum
*10	3rd portion duodenum		Severe peritonitis with perforated bowel

*Patients intubated during surgery.

Of the patients studied, there were ten in whom other methods of intubation failed. In these patients the average time of intubation with the stylet technique was twelve minutes. The over-all average time required for intubation was ten minutes. The shortest time was two minutes, and the longest, thirty minutes (Figs. 3 and 4).

Fig. 2.—R. F., 56-year-old white man. Moderate small bowel obstruction caused by adhesions. The tube and stylet are shown with the tip lying in the region of the ligament of Treitz.

Fig. 3.

Fig. 4.

Fig. 3.—J. A., 73-year-old white man. Previous gastric resection. The mercury bag of the Wild tube is seen in the gastric pouch, where it remained for fifteen hours.

Fig. 4.—Same patient as shown in Fig. 3. The tube and stylet are shown to be well down into the jejunum after five minutes of manipulation.

Two of the patients who were intubated had previous gastric resections. Other methods of intubation had been unsuccessful in these cases.

There were three patients in whom the intestinal tube was passed during surgery. The surgeon's hand guided the tip into the pylorus, and then the tube and stylet were pushed through into the jejunum.

The patients intubated with the stylet technique in whom there had been no previous attempts at intubation were those in whom some difficulty was foreseen, or in whom the urgent need for a tube in place called for a rapid and accurate method of intubation of the small bowel.

In several interesting cases the patients were intubated for clinical studies: A tube was placed in the proximal loop of a gastric resection eight days postoperatively; a tube was placed in the second portion of the duodenum in a patient who had a vagotomy ten days previously; the small bowel was intubated in two minutes in a patient who had a cholecystoduodenostomy.

SUMMARY

1. A new method of intestinal intubation is described and twenty-three cases in which it has been used are reported.

2. A flexible stylet with a controllable tip and a new long intestinal tube are described.

ADDENDUM

At the present time stylets and tubes are being built for use in treating children with intestinal obstruction. These will be used for intubation of patients at the fluoroscopy table and at the time of surgery. These will be smaller than the present stylet and tube and will be constructed very similarly to the one just described.

The authors wish to express their appreciation to Professor Owen H. Wangensteen for his active interest in this study. Dr. John J. Wild, Mr. Erich Rosendahl, and Mr. Edmund Hahn are thanked for their technical advice.

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DO CANCER PATIENTS WANT TO BE TOLD?

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THE problem of whether or not to tell a patient that he has cancer is an old one that must be met from day to day by the practicing physician. It frequently involves philosophic and psychologic considerations of extreme complexity. As may be expected, there are many viewpoints on this subject, varying from one extreme to the other.

Cancer is a disease which formerly by its very name carried the knell of doom to the victim. This dates from earlier days when there was little in the way of treatment to offer the victims of this disease. Now with the tremendous advances in diagnosis and treatment the situation is far from hopeless in many instances and does not justify the feeling of doom it formerly engendered.

Extensive efforts are being made to educate the public both in regard to the early recognition of the disease and to the benefits of timely treatment. These efforts are being expanded constantly so that more and more people are being made aware of the symptoms as well as the likely treatment of cancer. In view of this, it is difficult to believe that many people are not aware of the presence of cancer following the extensive examinations and treatment they receive.

Seelig[2] felt that the patient should not be told that his disease was cancer except in those uncommon instances in which special circumstances were present, or in which his cooperation could be won only by telling him the nature of the disease. He stressed the great mental shock of this sudden knowledge to the patient who associated it with the older concepts of the disease. However, he stated that his opinion might change if many of the popular concepts regarding cancer were changed. Sachs[3] said, "Do not tell a patient all you know or all you think you know." He also emphasized the psychologic trauma to the patient on learning his situation. Piatt[4] believed that each patient should be handled individually, depending upon his temperament. He felt that it is best not to tell the patient if the situation is hopeless unless special circumstances prevail. Solomon[5] also stressed the psychic effects on the patient and urged that the accent in education be placed on what can be done for the cancer patient at the present time, and not on the fearsome aspects of the disease, the statistics, or what the future may hold.

Cabot, however, believed the patient should be told the whole truth, with neither evasions nor withholding. Lund[6] subscribed to the precept, "As far as possible, do no harm." He points out that you can do harm either by telling

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the truth or by lying. Underwood[7] called fear, delay, and ignorance the allies of cancer and stressed the importance of education in the fight against the disease.

How does the patient feel about this problem? In an effort to obtain information on this point, a survey which was essentially an opinion poll was set up. Two groups of patients were surveyed, each group composed of 100 cases. One group consisted of patients in whom the diagnosis of cancer had been established. The other group was constituted of patients without known cancer. All of these patients were seen in the outpatient departments of the University Hospitals. The patients in the cancer group were all personally interviewed and their opinions recorded on the sheets prepared for the study. This group included both patients with operable and patients with inoperable or recurrent lesions, although the former were in the majority. The patients in the noncancer group were interviewed personally in some cases, and in others were simply asked to fill in the questions on the sheet handed them, although they usually were queried as to whether or not they understood what was asked when the forms were collected. In the latter group it was made clear this was merely an opinion poll and did not imply that they had cancer.

The following questions were asked of those patients in the cancer group. The method of querying in Question 1 was indirect at first, and as tactful as possible.

1. Is this the first time you knew about having cancer? Yes——— No———.

2. If you knew previously, when and how did you learn?

3. Doctors and patients' relatives are occasionally inclined to believe they are protecting patients from worry by not telling them they have cancer. Do you agree? Yes——— No———.

4. Patients with cancer are frequently cured by treatment but never find out they had cancer. It would probably help in the fight against cancer if these people knew their condition and could help to alert others to the possibility of cancer as well as offer encouragement about having it cured. Do you agree? Yes——— No———.

The noncancer patients were asked two questions:

1. If our examinations should disclose that you have a cancer, would you want to know about it? Yes——— No———.

2. Patients with cancer are frequently cured by treatment but never find out they had cancer. It would probably help in the fight against cancer if these people knew their condition and could help to alert others to the possibility of cancer as well as offer encouragement about having it cured. Do you agree? Yes——— No ———.

ANSWERS OBTAINED

In answering Question 3 of the cancer group queries, almost all the patients answered in the affirmative or negative and went on to say that they did or did not want to be told about having cancer. In addition, many of them tended to make a distinction between their own feelings on the matter and what they thought people in general would feel. In those who did not make this division spontaneously, specific questions were asked to elicit their opinion.

Cancer Group

Question 1.

85—Had previous knowledge of cancer.
15—Did not.

Question 2.

40—Were told by local physician.
33—Were told at the University Hospitals.
5—Were told accidentally by friends, etc.
7—Were told at another hospital.

Question 3. (A) In Regard to Self:

89—Preferred to know about having cancer.
6—Rather not.
5—Indefinite.

(B) In Regard to Telling People in General:

73—Think people should be told.
4—Think people should not be told.
3—Indefinite.
20—Believe it is an individual matter.

Question 4.

94—Agree.
3—Disagree.
3—Indefinite.

Noncancer Group

Question 1.

82—Want to be told.
14—Do not want to be told.
4—Indefinite.

Question 2.

88—Agree.
3—Disagree.
9—Indefinite.

Another group, consisting of patients being examined in the Cancer Detection Center at the University Hospitals, has been given the same questionnaire as was given to the noncancer group in this study. These patients were individuals over 45 years of age who were apparently well and on whom yearly complete physical examinations are being carried out in an effort to detect malignancies in an early stage.

To date, 740 opinions have been obtained in this group. The results are as follows:

Question 1.

729 (98.5%)—Want to be told.
7—Do not want to be told.
4—Indefinite.

Question 2.

737—Agree.
0—Disagree.
3—Indefinite.

Of the seven patients who answered no to Question 1, six of these specified that this was in the event that their condition was hopeless. It is probably to be expected that this group would be strongly in favor of being told about the presence of cancer, as cancer detection is the object of their undergoing yearly examinations on a voluntary basis.

DISCUSSION

It is interesting to note that whereas 89 per cent of the cancer patients said they preferred knowing their condition, of this same group only 73 per cent thought that people in general should be uniformly told. This would seem to imply that they feel others somewhat less capable of bearing the truth or more prone to emotional shock than themselves. Another interesting point is in regard to the wording of Question 3 in the cancer patients' form, which would tend to suggest a yes answer to the patient, whereas the answers obtained were almost overwhelmingly no.

In addition, twenty-four patients in the cancer group were asked if the knowledge that they had cancer had in any way altered their routine of existence or style of living outside of any physical disability. The answer was no in all cases.

Many of the patients remarked that they would be more willing and careful to follow directions in regard to necessary follow-up examinations if they knew they had cancer. Moreover, many of them stated that they worried more about the unknown and felt they preferred to know they had cancer, even if it was bad news, because it removed the indefiniteness of the situation. Some were indignant that they had not been informed sooner than they were. Of the fifteen patients who said they had not been previously told, most of these added that they had suspected their condition to be cancer. The fact that the remaining eighty-five patients in the cancer group already knew they had cancer would indicate that many physicians, both here in the hospital and in the local communities, are following the policy of informing the patients when they have cancer. The last question in both groups tends somewhat to confirm the answers given to the previous question as to whether or not the patient thought people should be told they have cancer. In addition it indicates that they believe in educating people in general to the possibility of cancer.

In an effort to see whether the opinions given by the patients have changed with the passage of time (approximately one year since the original study), we recently sent out letters to forty-one of the patients in the cancer group, asking them if their feelings were still the same in regard to whether or not they wanted to know about the presence of cancer. Answers were obtained in thirty-one cases. Two of the patients had expired in the interim. The remaining twenty-nine all replied essentially in the same way as they had on the first querying. This group included both those patients who originally stated they would rather not know about the presence of cancer as well as those who wanted to know.

Obviously the significance or validity of the replies obtained can be debated at great length. However, it would seem to indicate that patients want to be informed of the nature of their illness perhaps to a far greater extent than the average physician would anticipate.

SUMMARY

Two groups, each containing 100 individuals, were polled regarding the question of whether or not they would want to be told about having cancer and their answers were recorded. One group contained patients with known cancer and the other consisted of patients not known to have cancer.

The answers obtained would indicate that the great majority in both groups want to be told about the presence of cancer.

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AN EXPANDED INTERSTITIAL (THIOCYANATE) SPACE IN SURGICAL PATIENTS

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INTRODUCTION

THE concept that the body consists of an intracellular compartment surrounded by an extracellular fluid environment has been well established.[1] The maintenance of this organization is dependent largely upon the ability of the cell to permit a selective influx of certain ions and prevent permeation of others.[1, 2, 3] Chloride and sodium are considered to be the principal electrolytes of the extracellular space under ordinary circumstances, although in certain states they may enter the cellular structure.[3, 4, 5] As thiocyanate theoretically is distributed throughout the body as is chloride, the volume of the extracellular space ordinarily may be determined by measuring the dilution of a given dose of thiocyanate.[6, 7]

During the course of studies upon alterations of water and solutes in surgical patients, five patients were encountered who manifested marked increases in their thiocyanate spaces postoperatively. Concomitantly these patients exhibited complicated postoperative courses associated with the finding of an expanded extracellular space. The increase in the extracellular compartment could not be explained exclusively on the basis of administered fluid, and therefore it must represent either an influx of fluid from the intercellular compartment into the interstitial space, or an increased permeability of the cell membrane to the thiocyanate ion, and supposedly to chloride and sodium also.

Studies performed to identify the abnormality and the courses of the patients with expanded thiocyanate spaces form the subject of the present communication.

CHEMICAL METHODS

Serum chloride concentration was performed by the technique of Schales and Schales.[8] Serum proteins were determined by the method of Weichselbaum,[9] serum sodium by means of an internally compensated Perkin-Elmer flame photometer, the hematocrit as described by Musser and Wintrobe,[10] and carbon-dioxide combining capacity by the gasometric method of Van Slyke and Cullen.[11] Urine chloride concentration was ascertained by the modified Volhard-Harvey titration method[11] and is expressed in the tables as sodium chloride.

Plasma volume and thiocyanate (available fluid) space were measured by the procedure developed by Gregersen and Stewart,[12] adapted for the Evelyn colorimeter. The plasma volume was subtracted from the thiocyanate (extracellular space) to determine the interstitial space. One postinjection sample was obtained because Noble and Gregersen have demonstrated that results so

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TABLE I. J. J., F., 71; GASTRIC CANCER, SUBTOTAL GASTRECTOMY;

		INTAKE				OUTPUT				
DAY	WEIGHT (KG.)	TOTAL INTAKE (ML.)	BLOOD; PLASMA (ML.)	SODIUM CHLORIDE (GM.)	KCL (GM.)	TOTAL (ML.)	URINE VOLUME (ML.)	URINE SPECIFIC GRAVITY	CL IN URINE (GM. PER L.)	GASTRIC ASPIRATION (ML.)
Preop.	52.3	2750				900				
Surgery	51.1	3700	500b			1650	1450	1.021	1.0	200
1st day P.O.	51.2	2500				1850	1200	1.024	3.3	650
2nd day P.O.	52.4	2100				1400	850	1.021	3.0	550
3rd day P.O.	53.2	2200				2700	2000	1.011	3.7	700
4th day P.O.	51.2	2000	500p			2800	1325	1.005	0.8	475
5th day P.O.	49.6	3150		9		1920	1020	1.020	0.2	900
6th day P.O.	50.6	1800		9	1	2050	1330	1.006	1.9	720
7th day P.O.	48.8	3200	500p	6.5	1	2400	1920		4.8	480
8th day P.O.	49.5	3500		6.5	1	2325	1675			650
9th day P.O.	49.0	2550				1100	1100			

*A.M.
†P.M.

obtained vary but little from the method of studying several blood samples and projecting the dilution value to zero time.[13] Equilibrium in thiocyanate distribution is reached in twenty-five to thirty minutes,[14] and sampling at twenty minutes may give a slightly higher reading than that obtained by the multiple sampling technique, but the use of this method permits evaluation of differences within the same patient under conditions which could not affect mixing time.

CLINICAL OBSERVATIONS

The course of each patient is presented separately.

Expanded Interstitial Space Associated With Convulsions and Coma

CASE 1.—J. J., a 71-year-old white woman, was admitted to the University Hospital on July 24, 1949, with a diagnosis of adenocarcinoma of the stomach, left hydronephrosis, and cholelithiasis. She presented a past history of mild parkinsonism of six years' duration for which she had never received therapy. There was no history of encephalitis and no other central nervous system symptoms.

On admission the blood pressure was 140 mm. of mercury systolic and 90 diastolic. The heart was of normal size, and electrocardiogram tracing was normal. The venous pressure was 8 cm. of sodium citrate, and vital capacity equalled 2.4 L. A phenosulfonphthalein kidney excretion study revealed 76 per cent excretion 120 minutes after injection, and 44 per cent excretion 30 minutes subsequent to its administration.

The laboratory data are presented in Table I.

On July 28, 1949, the patient was subjected to a subtotal gastrectomy, left nephrectomy, and cholecystectomy under gas-oxygen-ether anesthesia. The operation lasted 4.5 hours, and during this interval she received 500 ml. of whole blood and 700 ml. of 5 per

HYDRONEPHROSIS, NEPHRECTOMY; CHOLELITHIASIS, CHOLECYSTECTOMY

BLOOD CHEMISTRY											
HGB. (GM. %)	HEMATOCRIT (%)	TOTAL SERUM PROTEIN (GM. %)	SERUM CL (MEQ. PER L.)	CO_2 COMBINING POWER (MEQ. PER L.)	NA (MEQ. PER L.)	K (MEQ. PER L.)	B.U.N. (MG. %)	BLOOD VOLUME (L.)	PLASMA VOLUME (L.)	INTERSTITIAL SPACE (L.)	REMARKS
13.5		5.3	106	22			10				
12.9	43.1	4.6* 5.0†	102 108					4.1	2.3	8.1	
13.2 11.8	43.7		83	17			7	5.0	2.9	15.6	Convulsions
13.6	43.5		82		113	3.9					Coma
	42.4		76				7.5				Coma
	45.5	4.8	82	26				6.2	3.6	9.6	Coma
12.9	42.5	5.0	89	28	146	3.4					Coma
12.2	38.5		91		150						Coma
											Coma
12.9	40.0	4.9	90	30							Coma
13.3	42.5	5.2	98	33			16				

cent glucose in distilled water. During the remainder of the day she received 3,000 c.c. of 5 per cent glucose in distilled water. The urine output for the day of surgery was 1,450 ml.

The immediate postoperative course was satisfactory. On the evening of the first postoperative day she suddenly developed a generalized tonic spasm followed by generalized clonic convulsions which lasted for about one-half minute. No respirations were observed during this period and she became cyanotic. Preceding the convulsive seizure the patient had been lethargic and the convulsions occurred shortly after attempts to arouse her. Immediately after the convulsions she became entirely unresponsive. The blood pressure remained 120 systolic and 60 diastolic, and the pulse rate continued at 60 beats per minute and became irregular. The skin was cold and clammy; Hoffmann's sign was negative, but she developed bilateral positive Babinski signs. The left eye deviated externally and the pupils reacted to light and accommodation. The abdomen was soft. The knee jerk and ankle jerk reflexes were absent bilaterally. The patient developed a second generalized grand mal type seizure about two hours after the first one, after which she became completely areflexic. A lumbar puncture performed on the second postoperative day revealed a pressure of 140 mm. of water. The protein content equalled 2 Gm. per cent, glucose 77 mg. per 100 c.c., and chlorides of 104 meq. per liter.

The serum chloride, which had been 108 meq. per liter during the immediate postoperative period, descended to 83 meq. per liter at the time of the first convulsive seizure. At this time the serum carbon dioxide combining power equalled 17 meq. per liter. On the second postoperative day the serum sodium was 113 meq. per liter and potassium 3.9 meq. per liter. The exact interpretation of the situation was uncertain, but it was believed by some to be on the basis of a cerebral vascular accident.

A rather interesting observation was the alteration in the thiocyanate (interstitial) space. Immediately after operation the plasma volume was 2.3 L., and the interstitial space 8.1 L. On the first postoperative day (before the onset of convulsions) the plasma volume had increased slightly to 2.9 L., but the interstitial volume had practically doubled

TABLE II. A. L., M., 82; GASTROJEJUNAL

		INTAKE			OUTPUT				
DAY	WEIGHT (KG.)	TOTAL INTAKE (ML.)	BLOOD PLASMA (ML.)	SODIUM CHLORIDE (GM.)	TOTAL (ML.)	URINE VOLUME (ML.)	URINE SPECIFIC GRAVITY	CL IN URINE (GM. PER L.)	GASTRIC ASPIRATION (ML.)
Preop.	40.6	1750			900				
Surgery	41.2 40.0	4200			650	550	1.018		100
1st day P.O.	43.2	3100			1700	1000	1.020	4.6	700
2nd day P.O.	42.0	1550	500b		2100	1650	1.024	5.1	450
3rd day P.O.	42.0	3500	1000b	13.5	1900	1450	1.016	1.0	450
4th day P.O.	43.4	2500			2900	2700		1.0	200
5th day P.O.	41.0	3175		4.5	2600	2600			
6th day P.O.					2500	2500		1.8	
7th day P.O.					1500	1500		1.0	

to the high value of 15.6 L. This increment of 7.5 L., not entirely the result of administered fluid, indicates that either an influx of intracellular water into the interstitial spaces had occurred or that the cellular membrane impermeability to thiocyanate had been disturbed.

The patient was treated with hypertonic glucose solutions intravenously, which induced a diuresis. On the morning of the fourth postoperative day the plasma volume was 3.6 L., and the interstitial space volume had diminished to 9.6 L. She became responsive, manifested no residual central nervous system disturbance, and on the ninth day she began to eat normally and was discharged on the fourteenth postoperative day.

CASE 2.—A. L., an 82-year-old white man, was admitted to the University Hospital on July 18, 1949, with a diagnosis of gastrojejunal ulcer following a gastroenterostomy twenty-five years before admission. Physical examination revealed the patient to be well preserved for his age. The blood pressure was 112 mm. of mercury systolic and 72 diastolic. The pulse rate was 72 per minute, and no murmurs were noted. The electrocardiogram demonstrated left axis deviation with an elevated ST_3 and a negative TCF_4. The laboratory data are presented in Table II. The white blood count was 5,100 cells per c.mm. with 46 per cent neutrophils. Gastric analysis revealed free acid, and stool examinations showed a positive (1 plus to 4 plus) reaction to benzidine. The Wassermann reaction was negative. The venous pressure was 9 cm. sodium citrate, and the vital capacity was 2.6 L. Liver function tests revealed a prompt reacting bilirubin to be 0.1 mg. per cent, and the total bilirubin was 0.4 mg. per cent, the thymol turbidity test was 2 units, and the cephalin cholesterol was 0. Serum alkaline phosphatase equalled 2.3 Bodansky units and the bromsulfalein test revealed 6 per cent retention in 45 minutes. The total serum cholesterol equalled 170 mg. per cent and the cholesterol ester was 116 mg. per cent.

Preoperatively the patient was maintained on a high protein diet, and received one blood transfusion of 500 ml. of whole blood.

On Aug. 3, 1949, he was operated upon under gas-oxygen-ether anesthesia. The operation, which lasted three hours, consisted of excision of the old stoma and a 75 per cent gastric resection. The estimated blood lost was 86 ml., and the patient received 1,000 ml. of 5 per cent glucose in distilled water during the operation. There was no significant drop

ULCER, SUBTOTAL GASTRECTOMY

BLOOD CHEMISTRY											
HGB. (GM. %)	HEMATOCRIT (%)	TOTAL SERUM PROTEIN (GM. %)	SERUM CL (MEQ. PER L.)	CO_2 COMBINING POWER (MEQ. PER L.)	NA (MEQ. PER L.)	K (MEQ. PER L.)	B.U.N. (MG. %)	BLOOD VOLUME (L.)	PLASMA VOLUME (L.)	INTERSTITIAL SPACE (L.)	REMARKS
13.5	43.5										
13.4	43.5	7.6	106	27				6.5	3.7	7.4	
		7.0	100								
13.5	40.0	7.1	92	27				4.5	2.7	12.9	Convulsions
11.7								4.0	2.4	14.2	
12.0	39.0	6.5	80	22	122		16				Coma
12.0			78				15				Coma
12.0											Coma
14.6	45.0	5.7	89	28	148		18				Coma
	49.0		90	31	152	3.6					Coma

in the blood pressure during surgery, and the patient left the operating room in good condition. When he returned to the recovery room the blood pressure dropped to 60/30, the pulse rate was 60 beats per minute, and the respiration rate was 16 per minute. He responded well to 1 ml. of Neosynephrin and the blood pressure rose to 130/70. The preoperative weight was 41.2 kg. and the postoperative weight was 40 kg. The removed tissue weighed 135 Gm. The patient was given, in addition to the quantity of fluids just mentioned, 3,200 ml. of 5 per cent dextrose and distilled water, and during the day of surgery he excreted but 550 c.c. of urine. The immediate postoperative course was uneventful, but on the morning of the first postoperative day he became somewhat apprehensive, a sensation which was followed by irregular jerkings of the limbs and a depression of the level of consciousness. Shortly thereafter he developed two generalized convulsions and became entirely unresponsive except to extreme stimuli. He maintained the irregular myoclonus involving principally the upper extremities. The eyes were fixed; the pupils were dilated, but responded to light and accommodation. Fundic examination revealed arteriosclerosis and no papilledema. The neck resisted flexion. No Trousseau sign was observed and no localizing signs were elicited. The blood pressure remained at 116/56, pulse rate 84 beats per minute, respiration rate 16, but the temperature increased to 103.2° F. and continued to spike this level for the next two days. The patient's weight increased to 43.2 kg. from a preoperative value of 41.2 kg. On the second postoperative day the serum proteins dropped to 6.5 Gm. per cent, serum chlorides to 80 meq. per liter, the carbon dioxide combining power to 22 meq. per liter, and serum sodium was 122 meq. per liter (Table II). The urine specific gravity fluctuated between 1.018 and 1.020, and the chloride concentration was 87 meq. of chloride per liter of urine.

The diagnosis was uncertain. It was believed by the neurological consultant to represent either a cerebral vascular accident or possibly cerebral anoxia. The convulsions were controlled by 7½ Gm. of Sodium Amytal. Spinal puncture performed the first postoperative day revealed the pressure to be 90 mm. initially. The spinal fluid contained 41 mg. per cent protein, 98 Gm. per cent sugar, and 98 meq. chloride. The colloidal gold test was negative. The culture of the fluid was sterile.

The plasma volume performed immediately postoperatively equalled 3.7 L. and the interstitial space was 7.4 L. The subsequent day (just preceding the convulsion) the

TABLE III. (CASE 3) E. O., F., 59; LIVER METASTASES FROM CARCINOMA

DAY	WEIGHT (KG.)	INTAKE (ML.)	OUTPUT		
			URINE VOLUME (ML.)	GASTRIC ASPIRATION (ML.)	CL OUTPUT (GM.)
Preop.	61.2	3150	1050		
Day of surgery	61.4	4000	800	100	1.2
1st day P.O.	61.8	3500	2200	400	5.5
2nd day P.O.	60.8	3600	1600	300	1.3
3rd day P.O.	60.6	3700	2500		

plasma volume revealed a decrease to 2.7 L. and the interstitial space increased to 12.9 L. That evening during a convulsive seizure the plasma volume equalled 2.4 L. and the interstitial space 14.2 L.

The patient was given 1,400 c.c. of 20 per cent glucose in distilled water intravenously on the first postoperative day, and 500 c.c. of 20 per cent glucose and 50 c.c. of 50 per cent glucose on the second day after surgery. On the third postoperative day the patient was given 1 L. of whole blood and 13½ Gm. of sodium chloride. He improved clinically with restoration of the blood chemistry (Table II). He was discharged on the eighth postoperative day with no residual manifestations of the previous convulsive and comatose state.

Increased Interstitial Compartment With Minimal Symptoms

CASE 3.—E. O., a 59-year-old white woman, was admitted to the University Hospital July 15, 1949, with complaints of eructations and weight loss of 25 pounds. She was a diabetic and had been taking 20 units of protamine zinc insulin each day. A segmental colectomy had been performed in June, 1943, for cancer of the transverse colon, and sub-

TABLE IV. (CASE 4) T. W., M., 38; CARCINOMA OF STOMACH.

DAY	WEIGHT (KG.)	INTAKE (ML.)	OUTPUT			
			URINE VOLUME (ML.)	GASTRIC ASPIRATION (ML.)	CL OUTPUT (GM.)	
					URINE	GASTRIC
8/1	52	4600	3000		3.6	
8/2	52	1600	1450		1.9	
8/3	52.2	1500				
8/4	51	3200	500		0.8	
8/5	52.2	3950	1500	250	2.4	1.2
8/6	51.8	3850	3500	600	3.8	3.0
8/7	50.8	3800	3500	900	4.5	4.5

OF TRANSVERSE COLON, DIABETES; EXPLORATORY LAPAROTOMY

BLOOD CHEMISTRY								
HGB. (GM. %)	HEMATOCRIT (%)	TOTAL SERUM PROTEIN (GM. %)	SERUM CL (MEQ. PER L.)	CO_2 COMBINING POWER (MEQ. PER L.)	BLOOD VOLUME (L.)	PLASMA VOLUME (L.)	INTERSTITIAL SPACE (L.)	REMARKS
13.4		6.8	97	28				
14.6	49.2	8.0	97		5.9	3.1	13.4	
	49.1	7.9	90	26	6.0	3.0	24.7	Poorly defined sensation of "ill feeling"
14.5	47.0							
	42.0		95	31				Profuse perspiration

sequently, she remained asymptomatic for five years, when she developed an upper abdominal mass. The heart was normal in size, and a soft, blowing, apical systolic murmur was audible. The blood pressure was 158/68. Laboratory data were essentially negative (Table III). The blood sugar was 83 Gm. per cent. Venous pressure was 8 cm. of sodium citrate and the vital capacity equalled 2.4 L. She excreted 47.5 per cent phenosulfonphthalein 90 minutes after its injection. Volume studies performed the morning of surgery revealed the blood volume to be 5.9 L., of which the plasma volume consisted of 3.1 L. The interstitial space was 13.4 L.

An exploratory laparotomy was performed under gas-oxygen-ether anesthesia, and hepatic metastases were encountered. Nothing further was done to the patient.

The afternoon of the first postoperative day, after she had received 4 L. of 5 per cent dextrose in distilled water and had urinated approximately 800 ml., volume studies revealed a precipitous increment of the interstitial volume to 24.7 L. (an increase of 11.3 L.). The blood and plasma volumes remained constant (Table III).

This case indicates that the expanded interstitial space can occur without necessarily producing the severe symptoms exhibited by the first two patients of this series. Her

SUBTOTAL GASTRECTOMY AND PARTIAL HEPATECTOMY

BLOOD CHEMISTRY									
HGB. (GM. %)	HEMATOCRIT (%)	TOTAL SERUM PROTEIN (GM. %)	SERUM CL (MEQ. PER L.)	CO_2 COMBINING POWER (MEQ. PER L.)	NA (MEQ. PER L.)	BLOOD VOLUME (L.)	PLASMA VOLUME (L.)	INTERSTITIAL SPACE (L.)	REMARKS
14.7	47.5		109	24		7.4	4.0	14.0	
13.7	46.0	8.3	106	25		8.1	4.4	12.9	
		8.2							
13.3	46.5		107			5.4	2.9	13.8	
12.5	40.0		90			7.1	4.3	22.9	
		7.6		27					Signs and symptoms of leak in gastrojejunostomy
	40.4		97						

TABLE V. (CASE 5) C. Z., M., 49; STOMAL

DAY	WEIGHT (KG.)	INTAKE			OUTPUT				
		FLUID (ML.)	BLOOD: PLASMA (ML.)	SODIUM CHLORIDE (GM.)	URINE VOLUME (ML.)	URINE SPECIFIC GRAVITY	GASTRIC ASPIRATION (ML.)	CL OUTPUT GM. URINE	CL OUTPUT GM. GASTRIC
Preop.	80	1000							
		900 (during surgery)							
½ hour P.O.	78.6	2800	500b		950		275		
1st day P.O.	78	3600	1500b		850	1.026	275		1.4
2nd day P.O.	81	2075		4.5	1175	1.027	200	1.9	1.0
3rd day P.O.	79	3200	500b	9.0	1750	1.028	350	3.3	1.8
4th day P.O.	79.6	3250	500b	9.0	3000	1.017		4.1	4.9
5th day P.O.	79.2	3485			1100			2.7	
6th day P.O.	77	2700			1200				

persistent complaint of a generalized sensation of "feeling ill" which she could not define may have been related to the altered fluid state.

Enlarged Thiocyanate Space Associated With a Leaking Gastroenterostomy

CASE 4.—T. W., a 38-year-old man, an Indian, was admitted to the University Hospital July 23, 1949, with a diagnosis of gastric cancer. The clinical findings were minimal on admission. The cardiovascular status was normal. The laboratory findings were essentially normal (Table IV). The serum bilirubin was 0.1 mg. per cent at 1 minute and 0.3 mg. per cent total. The thymol turbidity was 5 units and the cephalin cholesterol 1 +. The bromsulfalein excretion test revealed 1 per cent retention in 45 minutes. Total cholesterol was 164 mg. per cent and the cholesterol ester was 120 mg. per cent. The plasma volume was 4.0 L. and blood volume 7.4 L. The interstitial space averaged 14 L. Preoperatively the patient was given a test dose of fluid consisting of 4,600 ml. of 5 per cent dextrose in distilled water, which stimulated diuresis (Table IV). The subsequent day the patient's plasma volume was similar to that observed preceding the infusion and equalled 4.4 L. There was an associated slight diminution in the interstitial space equalling 12.9 L. His weight remained constant at 52 kg.

Three days after the water test, a subtotal gastrectomy and removal of hepatic metastases was effected, using gas-oxygen-ether anesthesia. On that day the plasma volume equalled 2.9 L. and the interstitial space was again 13.8 L. He was given 3,200 ml. of fluid on the day of surgery, plus 500 ml. of blood, and he excreted 500 ml. of urine. On the subsequent day he was given 3,950 ml. of 5 per cent dextrose in distilled water. The postoperative chemical alterations consisted of a drop of serum chloride from 107 to 90 meq., a drop in total serum protein from 8.2 to 7.6 Gm. per cent, and an increase in weight of 1.2 kg. Associated with these changes was an increase in the plasma volume to 4.3 L. and a sharp increase in the interstitial volume to reach the high level of 22.9 L.

On the third postoperative day the patient developed hematemeses associated with abdominal distention and rigidity. The pulse became rapid at 100 per minute, and the blood pressure dropped to 80/50. A diagnosis of peritonitis was entertained. On the seventh postoperative day he was re-explored and a small sealed leak in the Hofmeister

ULCER, SUBTOTAL GASTRECTOMY

BLOOD CHEMISTRY										
HGB. (GM. %)	HEMATO-CRIT (%)	TOTAL SERUM PROTEIN (GM. %)	SERUM CL (MEQ. PER L.)	CO_2 COMBINING POWER (MEQ. PER L.)	NA (MEQ. PER L.)	K (MEQ. PER L.)	BLOOD VOLUME (L.)	PLASMA VOLUME (L.)	INTERSTITIAL SPACE (L.)	REMARKS
18.4	51.5	7.0	101	28	143	3.9	6.7	3.3	17.7	
17.0	53.7	7.0	102	22	158.5	5.1	5.6	2.6	26.4	Shock
14.2	42.0									
14.3	44.2	6.6	96	27	140	5.0	4.6	2.6	17.4	
12.0	35.0	6.1	95	26						
10.0		5.9	95	30						
10.7	34.2									
12.1	38	6.7	95	28	144	3.3				

portion of the anastomosis found and repaired. He then made an uneventful recovery and was discharged on Aug. 23, 1949.

Enlarged Interstitial Phase Followed by Clinical Shock

CASE 5.—The patient, a 49-year-old white man, entered the University Hospital with a diagnosis of a gastrojejunal ulcer subsequent to a gastroenterostomy performed in 1937 for duodenal ulcer. Since that time he had had sporadic bouts of bleeding from the gastrointestinal tract.

On admission he appeared well nourished. The heart was normal in size and rhythm. No murmurs were audible. The blood pressure recorded 130 mm. of mercury systolic and 90 mm. diastolic. The pulse rate was 90 beats per minute. The admission blood chemistries were within normal limits (Table V). Tests for liver function revealed the thymol turbidity at 3 units, cephalin cholesterol negative. Bromsulfalein was 4 per cent retention at 45 minutes, serum bilirubin 0.1 mg. per cent at 1 minute and 0.6 mg. per cent total. Total serum cholesterol was 224 mg. per cent, of which the cholesterol ester composed 74 per cent. The alkaline phosphatase content of the serum was 1.0 Bodansky units.

Preoperatively the blood volume was 6.7 L., the plasma volume 3.3 L., and the interstitial space 17.7 L. The total body water as determined by deuterium dilution was 57.8 L. (72 per cent of the body weight).

A subtotal gastric resection was performed Sept. 13, 1949, utilizing gas-oxygen-ether anesthesia and morphine sulfate and hyoscine as the preanesthetic drugs. The estimated blood loss was 350 ml., and the lowest recorded blood pressure during the operation was 80/60. He received 900 ml. of 5 per cent dextrose in distilled water during the period of surgical intervention. Immediately after the operation, blood chemical studies were not significantly changed (Table V) except for an unexplained elevation in serum sodium.

However, an impressive increase of the interstitial volume from the preoperative value of 17.7 L. to 26.4 L. was noted immediately postoperatively. Associated with an increment of 8.7 L. in the interstitial space was a decrease in both the blood volume and plasma volume of 1.1 L. and 0.7 L. respectively. The total body water postoperatively averaged 56.4 L.

TABLE VI. URINARY EXCRETION OF ELECTROLYTES AND NITROGEN IN PATIENT 5 DURING AND TWENTY-FOUR HOURS SUBSEQUENT TO A PARTIAL GASTRECTOMY

		CL		NA		K		N	
	VOLUME (ML.)	CONCENTRATION (MEQ. PER L.)	TOTAL AMOUNT (MEQ.)	CONCENTRATION (MEQ. PER L.)	TOTAL AMOUNT (MEQ.)	CONCENTRATION (MEQ. PER L.)	TOTAL AMOUNT (GM.)	CONCENTRATION (GM. %)	TOTAL AMOUNT (GM.)
During surgery, 6.5 hours	950	75	71.3	72.8	69.2	34.2	32.5	3.4	3.2
Total 24 hour period P.O.	850	37	31.5	19.5	16.6	76.5	65.0	9.0	7.7

At the time these volume determinations were performed the blood pressure was 120 mm. systolic and 80 mm. diastolic. The pulse was of good quality and averaged 90 beats per minute. The first three postoperative hours were without event. He was given 500 mm. of whole blood and 1 L. of 5 per cent dextrose in distilled water during this time.

On the fourth postoperative hour the blood pressure declined to 82/60; the pulse rate increased to 120 per minute, and the respirations to 28 per minute. The patient suddenly became markedly restless, began to perspire very profusely, and the skin became cold and clammy. Ephedrine, 25 mg., and another liter of 5 per cent dextrose in water were administered, but the patient's condition did not improve. The blood pressure dropped to 65 millimeters systolic and the diastolic pressure was imperceptible. He was given several 50 mg. doses of ephedrine and 1,500 ml. of whole blood. Seven hours after surgery the hemoglobin was 14.2 Gm. per cent and the hematocrit was 42 per cent red blood cells. After 1 L. of whole blood, the blood pressure rose to 120 mm. systolic and 80 mm. diastolic.

On the first postoperative day the blood hemoglobin equalled 14.3 Gm. per cent, the red cell count was 4.2 million cells per c.mm. and the hematocrit equalled 44.2 per cent red cells. The volume studies at this time were of particular interest, for the blood volume had decreased 1 L. (average 4.6 L.) despite the administration of 2,000 ml. of whole blood, an amount greatly in excess of the 350 ml. blood loss at surgery. The plasma volume had not changed from that noted immediately postoperatively, and the interstitial volume had returned to its preoperative value. The hemoglobin value continued to diminish, and on the fourth postoperative day, after an additional liter of whole blood had been administered, the hemoglobin value remained low (10.7 Gm. per cent).

An explanation for the postoperative shock and anemia was wanting. Bleeding either into the peritoneal cavity or the gastrointestinal tract was considered as the most likely cause, although it could not be confirmed. The preoperative bleeding and clotting times were 3 minutes and 2 minutes 15 seconds respectively The patient subsequently made an uneventful postshock recovery and was discharged on the seventh postoperative day. Although it is possible that the shock and anemia may have been due to occult bleeding, since this circumstance was associated with a markedly expanded interstitial space this case is included.

The urinary excretion of the electrolytes of this patient is presented in Table VI, which reveals that during the period of surgery (at which time the expanded interstitial space developed) 32.5 meq. of potassium were excreted. During the subsequent twenty-four hours, 65 meq. were excreted. During these same time intervals, 3.2 and 7.7 Gm. of nitrogen respectively were identified in the urine. These values are similar to those observed during the postoperative period in patients who did not develop the expanded interstitial space, and signify that no unusual breakdown of protoplasm occurred and that no massive shift of intracellular fluid into the extracellular space took place. The data accordingly suggest an increased permeability of the cells to the thiocyanate, per-

mitting thereby an influx of that ion into the intracellular compartment. The data demonstrate further that the interstitial phase may alter its volume independently of changes in the plasma volume.

The possible relationship between the observed expanded interstitial space which preceded the onset of severe clinical shock invites interesting speculations, but the explanation must await further experimentation. The fact that it did occur suggests a relationship.

DISCUSSION

The five patients presented in this study manifested significant expansion of the interstitial space, usually to very marked degrees. Increases in extracellular compartments may occur following the administration of large volumes of isotonic solutions of sodium chloride,[18] or following heart failure,[19] eclampsia,[21] or the administration of 5 per cent dextrose in water if urinary excretion is prevented.[16, 20] The reason for the expanded interstitial spaces in the patients herein presented is not entirely manifest. The quantity of fluid administered to the patients of this series could not account for the degree of expansion noted. One patient (Patient 5) revealed a diminished body weight and total body water associated with the enlargement of the interstitial compartment. This finding demonstrates that the enlarged interstitial compartment was not the result of a gain of body fluids. In the other cases none showed a weight gain that would indicate an extracorporeal source of fluids.

An expanded interstitial volume could result from an influx of water either from the plasma or the intracellular compartment. The latter is known to occur following the intravenous administration of hypertonic salt solution.[22] In this series, although a decreased plasma volume was observed at times associated with the increase of interstitial fluid, the decrement of plasma was entirely too small to supply sufficient fluid necessary to produce the degree of expansion of the interstitial space. No hypertonic saline solutions were given these patients to help explain the phenomenon on the basis of influx of intracellular water in answer to osmotic demands. Large doses of 5 per cent dextrose in distilled water were given, and it has been demonstrated that this solution normally diminishes the volume of extracellular fluid.[18]

Furthermore, an enlarged extracellular space on the basis of fluid coming from the cells would result in an intracellular dehydration. The observation that two patients of this series suffered from convulsive seizures suggests an intracellular edema[15, 20] rather than intracellular dehydration.

Studies of potassium and nitrogen excretion performed on Patient 5 (this series) revealed a similar urinary potassium and nitrogen output to that of other surgical patients under the same conditions, but no unusual interstitial alterations were manifested. These findings suggest also that there was no large outpouring of intracellular water into the extracellular space.

These data accordingly suggest that the cellular membrane may have become permeable to the thiocyanate ion, permitting the ingress of that ion and water into the intracellular compartment. Since thiocyanate is believed to simulate chloride in its distribution,[6] it follows that chloride also has been permitted to transgress the cellular barrier. The thiocyanate space, therefore,

under these conditions, may not measure the extracellular compartment in its strict sense, for a segment of the intracellular compartment may be included in the measurement.

A decrease in the concentration of serum chloride which could not be accounted for by excretion suggests that a corporeal redistribution of the electrolyte which is usually situated in an extracellular location had occurred. Intracellular ingress of chloride and thiocyanate have been described in various conditions,[23, 24, 25] and its probable occurrence in the patients of this series implies a clinical significance in surgical patients.

Postoperative expanded extracellular spaces have been described by Lyon, Stanton, Freis, and Smithwick.[26] Analysis of their data revealed two patients who demonstrated an increase in the extracellular space of 8.9 and 11.5 L. respectively during their postoperative courses. Their data do not indicate that the increment resulted from administered fluid, and in all probability a phenomenon similar to that described in this presentation had occurred.

The cause for the enlarged interstitial space or abnormal distribution of thiocyanate is not discernible. Anesthesia[27, 28] or surgical trauma may be factors. Hopkirk and Cope[17] have described a similar phenomenon following ruptured duodenal ulcers, and Patient 4 of this series probably represents such an instance. It is of interest that preceding surgical intervention this patient's response to a load of 4 L. of 5 per cent dextrose and distilled water differed markedly from that manifested postoperatively (Table IV). Overman and associates[29] have described this condition in simians and human beings suffering from malaria.

Severe fluid translocations have been observed to occur as a result of hormonal imbalance. Gaudino and Levitt[30] have demonstrated in dogs that administration of desoxycorticosterone acetate results in a diminution of intracellular water with an increased extracellular space. The total body water and plasma volume was apparently not altered. These facts suggest the possibility that the fluid alterations observed in this series may possibly be due to a hormonal imbalance. These authors, moreover, demonstrate that the thiocyanate space is not a true index of the absolute extracellular compartment (measured as the inulin space), for it apparently transgresses cellular boundaries, which observation supports the premise resulting from this study that the expanded thiocyanate space probably represents an influx of thiocyanate into the intracellular compartment rather than a true expansion of the extracellular compartment.

The clinical behaviors of the patients who exhibited the distorted interstitial space were unusual. Two patients manifested severe convulsions followed by semicomatose states for a period varying from one to ten days. Since it has been demonstrated that water retention produces convulsions[15, 16, 20] and factors which contribute to water retention enhance convulsive episodes in epileptics, it is suggested that water retention contributed to the convulsive seizures in the patients of this series. The abnormal permeability possibly augmented the intracellular edema. The low serum chloride and the low spinal fluid chloride

concentration in these patients in the absence of abnormal chloride losses favors the hypothesis of an abnormal electrolyte distribution. One of the patients (Case 1) suffered from parkinsonism, and the altered central nervous system was apparently more sensitive to convulsive reactions from the stimuli.[16]

The nature of shock in Patient 5 observed five hours after the increased interstitial space was noted cannot be explained.

In two patients (Cases 4 and 5) in whom interstitial volume studies were performed subsequent to the enlargement of their interstitial space, the data revealed relatively normal values suggesting that the deformity was transient.

This report is of a preliminary nature, and studies using radioactive isotopes and inulin are contemplated to identify more exactly the abnormality and the association of alterations in the interstitial compartment and its application to clinical surgery.

SUMMARY AND CONCLUSION

1. Five patients are presented who manifested a marked expansion of their interstitial spaces, as measured by thiocyanate dilution, during their postoperative course.

2. The expanded thiocyanate space could not be due to fluid administration, and evidence is presented demonstrating that it represents an abnormal permeability of the cell to the thiocyanate ion.

3. Chloride and sodium are affected also in this mechanism, which accounts for the low values of the serum chloride concentrations in these patients.

4. The clinical implications of this phenomenon are discussed.

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THE FATE OF AN ARTERY IMPLANTED IN THE MYOCARDIUM

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ATTEMPTS at increasing the vascularity of the heart have centered in general about three types of procedures: (1) interruption of the nerve supply of the heart as illustrated by the work of Fauteaux[3]; (2) reversal of the flow of the coronary sinus with or without an arterial shunt such as Beck[1, 2] has produced; and (3) formation of adhesions with other structures, as illustrated by the report of Thompson[5] on pericardial adhesions and the omental grafts of O'Shaughnessy.[4]

Little seems to be known about the fate of a systemic artery placed in the myocardium and, except for the studies of Vineberg,[6, 7] we have been unable to find reports in the literature on this subject. Because of the paucity of information on this approach, we were stimulated to study the results of implanting the internal mammary artery into the myocardium of the left ventricle of dogs.

METHOD

Six mongrel dogs, male and female, weighing from 26 to 32 kg., were used in this series. The animals were anesthetized with intravenous Nembutal (0.2 c.c. per pound) and positive pressure insufflation was maintained through an endotracheal tube by means of a motor-driven respirator.

The steps in the operative procedure are illustrated in Figs. 1 to 3. The chest was opened through the left fourth interspace, and the left subclavian and internal mammary arteries were mobilized by ligating and dividing the branches of these vessels. A bulldog clamp was placed on the subclavian artery. The internal mammary artery was divided near the sternum and the distal end ligated. The proximal end of the artery was left open and a single mattress suture of 00000 arterial silk was placed through the slightly beveled open end of this artery in such a manner as not to occlude its lumen.

The pericardium was opened in a longitudinal direction and a traction suture of fine silk was placed through the apex of the heart. A long, hollow, curved No. 15 gauge needle was passed through the myocardium of the left ventricle to the left of the anterior descending branch of the left coronary artery, so that a tunnel approximately 2 cm. in length was created. A stylet was passed through the needle and the arterial silk suture attached to the internal mammary artery was tied to the stylet. The stylet was pulled up to a point at which the artery was snug against the hollow needle and the needle was gently pulled through the myocardium, the artery following it. The artery was allowed to retract until the open end was just beneath the surface of the epicardium. The arterial suture was tied to the traction suture that had been placed through the apex of the heart, to prevent further retraction.

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The bulldog clamp was removed from the subclavian artery and the operative site inspected for bleeding. Hemorrhage was not a problem in these experiments, although in two cases it was necessary to apply Gelfoam to the surface of the heart at the point of exit of the artery to control bleeding.

One of the six animals was sacrificed each week. An anesthetic dose of intravenous Nembutal was given and the chest opened. The animals were allowed to succumb by anoxia following observations of the operative field.

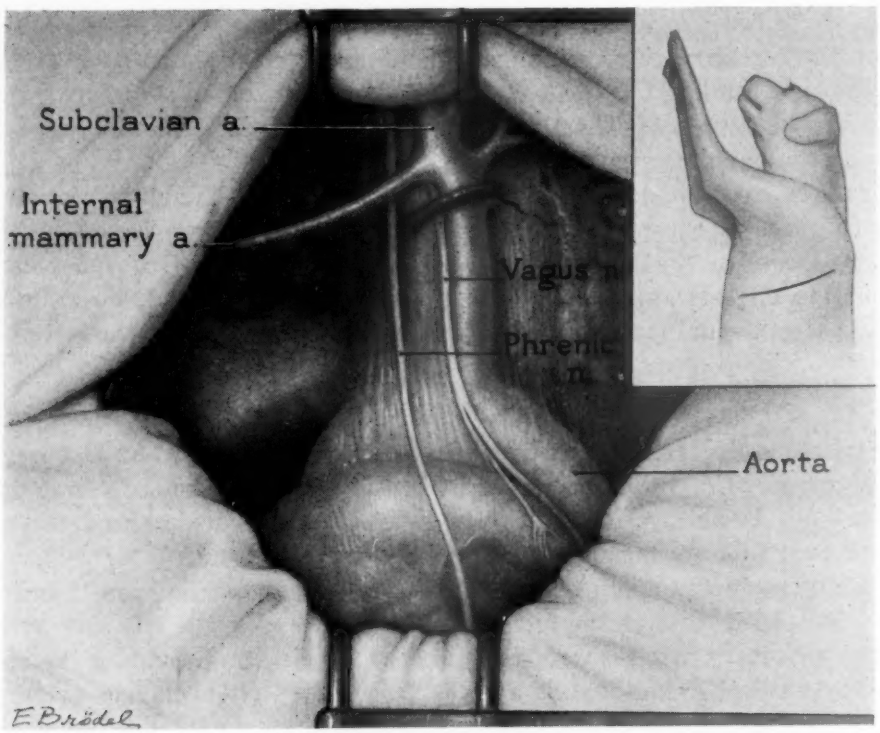

Fig. 1.—The anatomic position of the internal mammary artery and the subclavian artery is illustrated as viewed through the approach from the left pleural cavity. The position of the incision at the level of the left fourth interspace is diagrammatically represented in the inset in the upper right corner.

RESULTS

Gross Findings.—At the time of sacrifice, there were usually a few easily separated adhesions between the operative site and the lungs. The subclavian in five animals transmitted a readily palpable pulsation. In one dog the adhesions were sufficiently dense to prevent satisfactory palpation prior to cessation of cardiac activity. After the heart and great vessels were removed, the

Fig. 2.—The pleura has now been incised and reflected over the vessels and the branches of the subclavian artery ligated and divided. The internal mammary artery is tied distally and divided. The arterial silk suture is shown in position through the open end of the internal mammary artery. The bulldog clamp has been placed on the subclavian artery to control bleeding through the open end. The position of the curved needle in the myocardium of the left ventricle is illustrated with the long end of the suture from the internal mammary ready to be tied about the stylet.

Fig. 3.—The final step in the implantation of the internal mammary artery into the myocardium has been completed. The artery has been drawn into position by withdrawal of the curved needle and stylet shown in Fig. 2. The suture through the open end of the artery has been tied to the apical traction suture to maintain the position of the implanted artery.

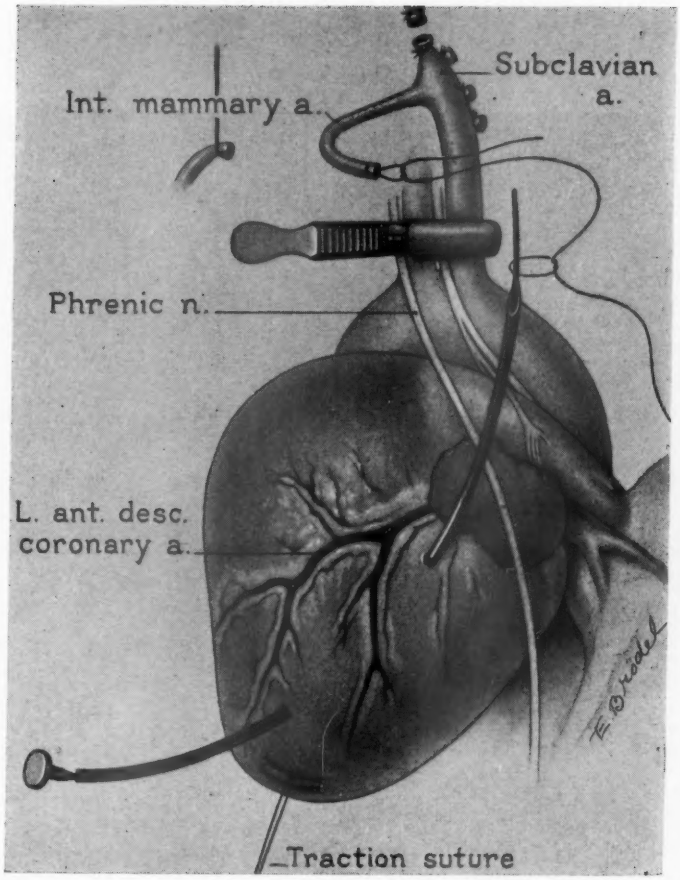

Fig. 2. *(For legend see opposite page.)*

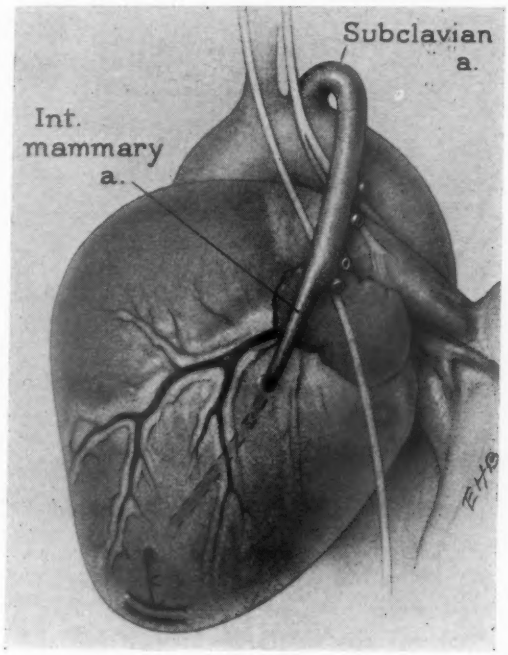

Fig. 3. *(For legend see opposite page.)*

Fig. 4.

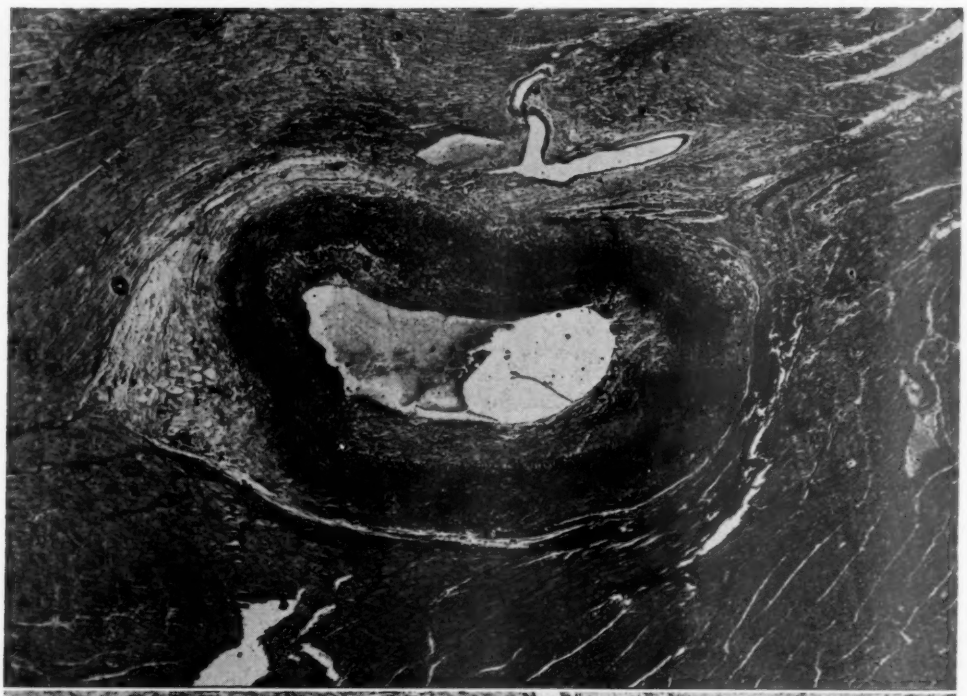

Fig. 5.

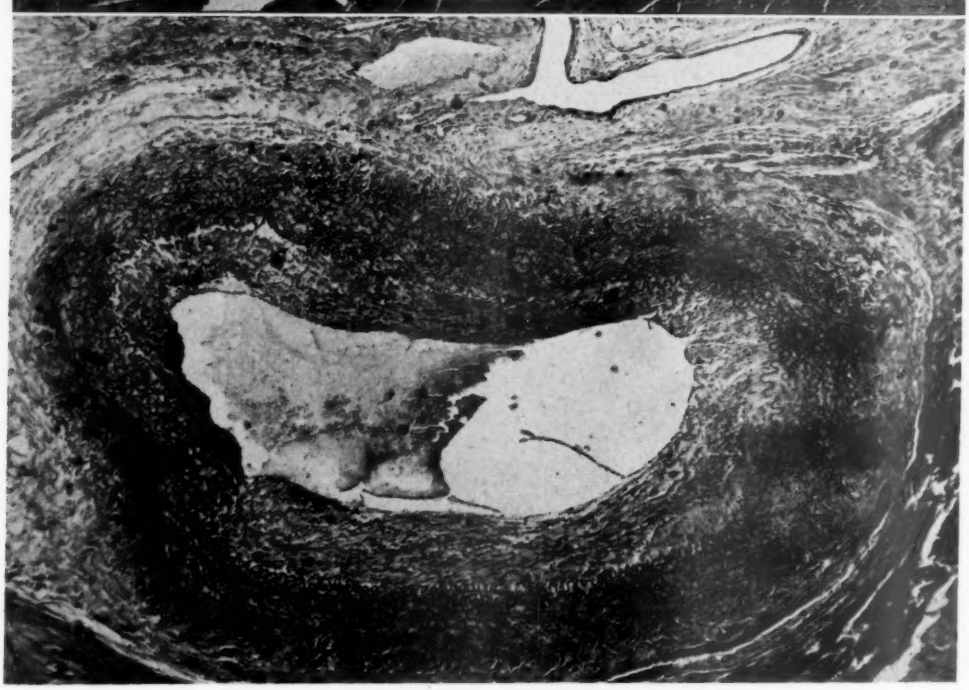

Fig. 4 (Dog 832).—Transverse section through the internal mammary artery six weeks after implantation in the myocardium of the left ventricle. This section is from the proximal portion of the buried artery (×40).

Fig. 5 (Dog 832).—Higher magnification of Fig. 4 (×80) to demonstrate the intimal proliferation and well-preserved media. The lack of reaction in the surrounding myocardium is apparent.

Fig. 6.

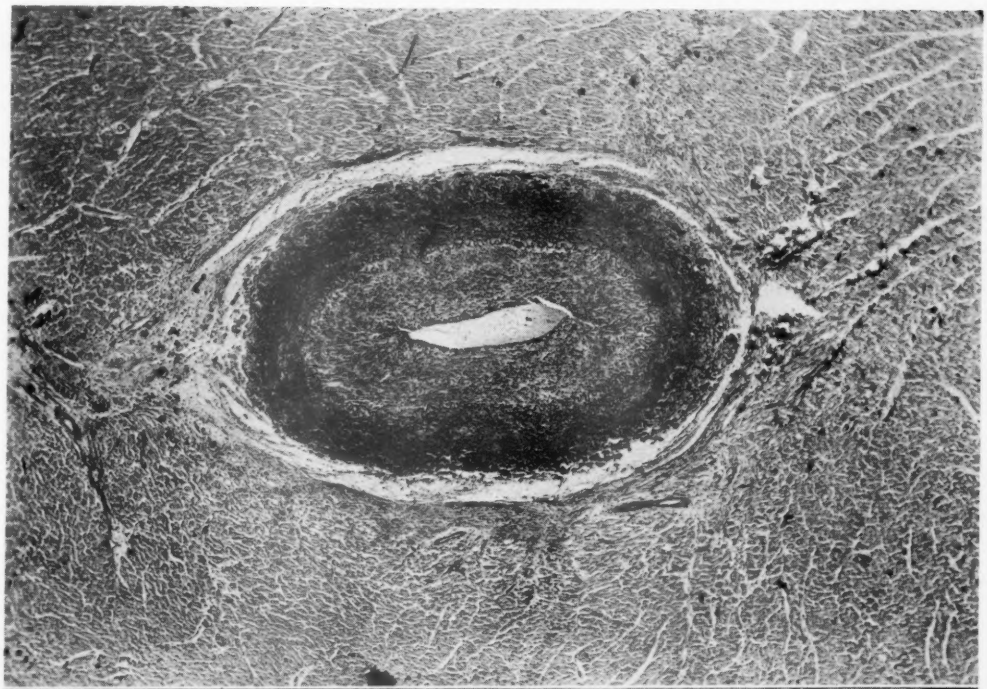

Fig. 7.

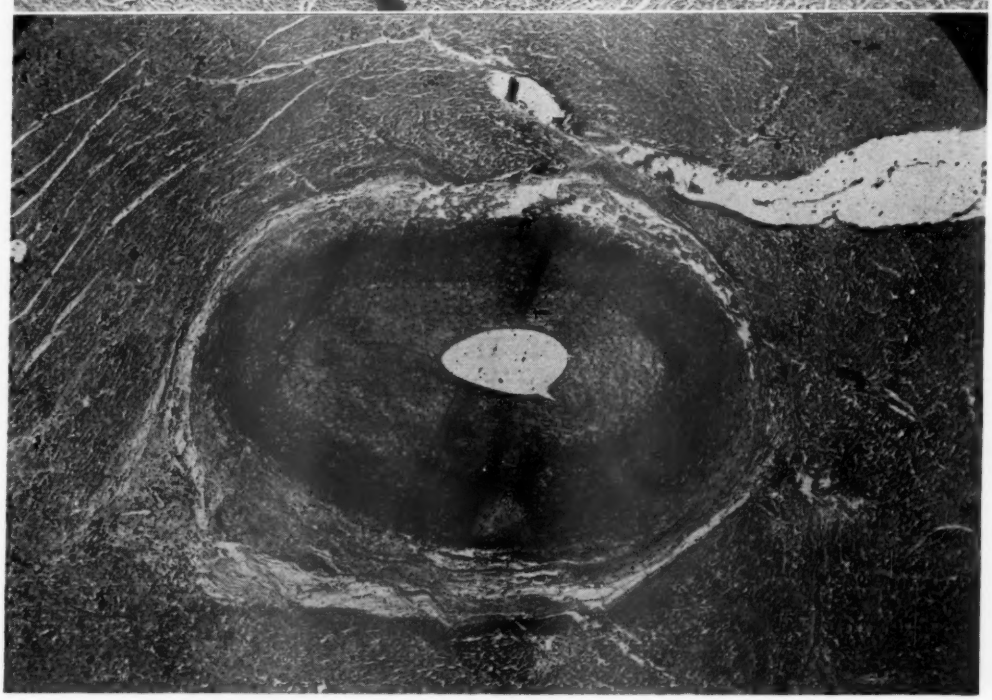

Fig. 6 (Dog 890).—Section through the mid-portion of the implanted artery, showing rather marked intimal proliferation. This animal was sacrificed three weeks after operation (×40).

Fig. 7 (Dog 885).—Section through the distal third of the internal mammary artery five weeks after implantation into the myocardium (×40).

subclavian and internal mammary arteries were opened from the origin at the aorta. All specimens were free of thrombus formation to the junction with the myocardium. The heart was opened and the endocardium inspected. There was no evidence of damage to the endocardium and no mural thrombus. The block of myocardium containing the implanted artery was removed and immediately placed in fixing solution.

Microscopic.—The striking microscopic finding has been an obliterative endarteritis (Figs. 4 and 5). In each instance an intimal proliferation was found, but in only one case was the obliteration of the lumen complete. In this specimen, the obliteration occurred at the site of entrance of the artery into

Fig. 8.—In this black and white photograph of the perfusion, the blue latex injected into the transplant shows up lighter than the red used in the remaining intact coronary circulation. The X marks the beginning of the transplant. The communications that have been established are to the left.

the myocardium and a patent lumen was found on either side of the occluded area. The myocardium appeared to tolerate the implanted vessel well (Figs. 6 and 7). Some hemosiderin was observed in the immediate vicinity of the vessel, particularly near the terminal portion, but there was little fibrosis. In the specimen obtained one week after implantation, small scattered deposits of calcium were found in the myocardium immediately adjacent to the vessel, but this was not found in other cases. It is possible that these calcium deposits were related to the trauma of the operative procedure of passing the artery through the artificial tunnel. The media was well preserved, as is illustrated in Fig. 6.

The terminal portion of the buried artery showed essentially two types of changes: (1) a foreign body reaction related to the arterial silk suture used to pull the artery into position and (2) a process of organization and canalization. There was no evidence of hematoma formation. There were small endothelial-lined channels coursing through the area at which the implanted artery terminated, and these seemed to have arisen from the artery rather than from the heart, when traced through serial section. We have perfused the intact coronary circulation and the transplant in sacrificed animals with liquid latex, using a red preparation for the coronary circulation and blue for the transplant. Fig. 8 indicates how functional such implants have been in one animal. These findings are of interest since bleeding from the implanted artery has not been a problem.

DISCUSSION

The experiments described are preliminary to additional work in progress at the present time and analysis of the results on this group is limited by the brevity of the period of observation and the small number of experiments performed.

Three interesting findings are illustrated in the photomicrographs. First, there is a lack of reaction in the myocardium to the implanted artery. The animals tolerated the operative procedure well and the sections have shown that the myocardium reacts little to the systemic artery buried within its substance. Second, the lumen of the implanted artery remains patent in spite of a rather marked intimal proliferation. Since this has persisted for as long as six weeks after operation and since small vessels have been found in the region of the junction of the artery and myocardium, it may be assumed that blood is circulating through the vessel. Further study is necessary to determine the volume, direction of flow, and the possible anastomosis with the coronary vessels. Third, the changes at the end of the implanted artery are those of organization and channeling. It was feared that placing the open end of a systemic artery in the myocardium might result in hematoma formation and fibrosis, but this has not been found. The eventual fate of the implanted artery is being investigated to determine the outcome of these changes.

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LIGATION OF LEFT AURICULAR APPENDAGE FOR RECURRENT EMBOLIZATION

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SUDDEN loss of life or limb due to embolic occlusion of a major artery is a challenging therapeutic problem. We have made great strides in the management of thrombosis of the leg veins in order that sudden pulmonary artery occlusion be averted. Ligation of a major venous channel from the leg, or use of anticoagulants, has helped materially in the management of this form of thrombosis and subsequent embolism. However, as yet relatively little has been done to help avert major embolization from mural thrombi of the heart.

The first major surgical attack upon one aspect of this problem was suggested to Madden[1] in 1946 by William Dock. It was reasoned that since in rheumatic mitral stenosis one of the most common origins of an embolus was as a mural thrombus within the left auricular appendix, a good approach to the problem of recurrent embolization in that disease would be to resect the auricular appendix. Accordingly, in 1948, Madden attempted this procedure in two patients, with the postoperative loss of one.

It occurred to us, at the time this presentation was made, that perhaps a simpler expedient to the problem of prevention of recurrent embolization would be merely to ligate the left auricular appendage at its junction with the left atrium. In preliminary experiments in dogs,[2] we found the auricular appendage actually fibrosing down almost to its disappearance in a matter of three months following placement of a simple ligature at its base. This procedure appealed to us much more than resection, because of its simplicity. In addition, the curvature of the atrium at the point of juncture is such that a clamp placed there is very likely to slip.

Hellerstein, Sinaiko, and Dolgin[3] and Burdette[4] have recently suggested experimental methods of resection of auricular appendages. Burdette made use of preplacement sutures which are tied, and then the auricle is resected. Whatever the procedure attempted, it is our belief and experience that rheumatic hearts that are to be operated upon do not stand much manipulation, or long anesthesia. It therefore behooves one to perform the simplest and most effective procedure and close the chest.

The three following case histories are presented as cases of rheumatic mitral stenosis with embolization, following which ligature of the left auricular appendage was performed.

CASE REPORTS

CASE 1.—J. B. (Ancker Hospital No. 28210), a 47-year-old white woman, was first seen for a cardiac condition on June 11, 1949. At that time she complained of dyspnea and orthopnea of six months' duration, and pain in the left flank of six days' duration. In

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December, 1948, this patient had an attack of severe chest pain that made her rather dyspneic. For two days after this attack, she had rather diffuse chest pain which eventually disappeared. Prior to December, 1948, she had had slight exertional dyspnea, but since that time she has had this continually. During the three months previous to admission she had a pain in the left flank that radiated around the abdomen. This pain was intermittent in character.

The patient's past history revealed no evidence of rheumatic fever, high blood pressure, or chorea. Blood pressure was 120/80, pulse 102 with fibrillation.

Physical examination revealed nothing of note except that the heart was enlarged to the left. Second aortic sound was weaker than the second pulmonic sound. There was a coarse systolic murmur at the apex and the base. Auricular fibrillation was rapid. The spleen was not enlarged, but the liver was down five fingerbreadths below the costal margin.

Electrocardiogram on June 12, 1949, revealed an abnormality with auricular fibrillation and probable left ventricle strain.

A chest x-ray examination revealed generalized cardiac enlargement with the configuration of the heart suggesting a mitral lesion. There was some suggestion of pulmonary congestion.

Results of laboratory studies were: urine normal; hemoglobin 8.8 Gm.; white blood count normal; blood cultures negative; and a sedimentation rate somewhat elevated. During the course in the hospital she had had some fever which was controlled with penicillin. The possibility of subacute bacterial endocarditis was considered. However, no direct proof could be obtained. A definite diagnosis of mitral stenosis and auricular fibrillation was made. On Salyrgan therapy, the patient lost about thirty pounds of edema fluid. During her course in the hospital she showed continued improvement for about two weeks. Against the advice of the physician in charge, she left the hospital.

She was seen again on Sept. 29, 1949. At that time the main complaint was a numbness and pain in the right leg. She had been getting along fairly well up to that time. The extremities revealed a dusky cyanosis of the right leg to about 8 cm. below the knee. There was no dorsalis pedis or posterior tibial pulse palpable. The leg was cold and there was a loss of sensation in it. Femoral pulse was palpable at the time. The impression was an arterial embolus at the bifurcation of superficial and deep femoral arteries.

Embolectomy was considered, and done* about five hours after the initial onset of pain. Under local anesthesia an embolus was removed from the common femoral artery; an excellent result was obtained. The patient made a completely uneventful recovery, and after consultation with the medical staff, it was decided that an auricular ligation should be attempted. On Oct. 24, 1949, auricular appendage ligation was performed on the left side through a third intercostal space incision. Intravenous 1 per cent procaine was kept going throughout the procedure. The pericardium was incised and the sac was entered. There were many pericardial adhesions present and these were dissected off the auricular appendage. The left atrium was quite enlarged and obviously under high pressure. A thin umbilical tape was used to ligate the junction of the auricular appendage and the left atrium. The left auricular appendage was felt and the presence of small thrombi could be ascertained. Pericardium was closed with 0000 silk leaving a small opening of about 2 cm. at the lower angle of the incision. The chest was then closed in the usual manner and the patient returned to the ward in good condition. Postoperative recovery was essentially uneventful and she was discharged Nov. 2, 1949. She was last seen on Dec. 13, 1949, at which time she had no complaints. Fibrillation is still present.†

CASE 2.—J. D. (Ancker Hospital No. 165329), a 40-year-old white man, was first admitted to the Ancker Hospital on Nov. 26, 1949. This man had been followed at another hospital and by a local physician for a period of time. At the age of 15, the patient apparently had contracted a case of rheumatic fever, with heart involvement in 1929. In 1943, he had an episode which suggested an embolus to the cerebral area with some weakness on the right side resulting. He had a similar episode again in 1949.

*By Dr. Frank Quattlebaum.

†This patient was again seen on Feb. 24, 1950, at which time she was in good condition.

Past history revealed essentially nothing and a physical examination showed blood pressure of 128/60 with an irregular pulse of 84. There was some evidence of retracted eye fields. Chest examination showed a displaced sternum diagnostic of pectus excavation. The second pulmonic sound was greater than the second aortic. There was a definite pulse deficit of 10. Diagnosis of rheumatic mitral stenosis with embolization had been made. The patient wished to be free of the possibility of any recurring emboli. It was suggested to him that an auricular appendage ligation be done.

Operation was performed on Nov. 28, 1949, under intratracheal anesthesia. Intravenous 1 per cent procaine was kept going throughout the procedure. The left side of the chest was opened through the third interspace and the pericardium approached. This was opened and the left auricular appendage came into view. It was quite tense and on palpation, a suggestion of two small thrombi were felt. The left atrium was under a tremendous amount of tension. A ligature of thin umbilical tape was then placed at the base of the left auricular appendage at its junction with the left atrium. The patient withstood this procedure well. The pericardium was closed leaving only a small opening at the lower angle. The chest was closed in the usual manner. Postoperative recovery was marred only by the questionable presence of an atelectasis of left lower lobe. However, on further fluoroscopic examination, this was excluded. He was discharged on Dec. 6, 1949, with fibrillation still present.*

At the time of this communication, the patient had no cardiac complaints, no dyspnea, chest pain, or peripheral edema.

CASE 3.—G. H. (Ancker Hospital No. 10585), a 56-year-old white woman, was admitted to the Ancker Hospital on Nov. 14, 1949. The main complaint at the time was sudden sharp pain in the right thigh and leg. During examination of the colon with a barium enema, she developed this sudden sharp pain in the right thigh and was soon seen by the medical service. On examination, a dyspneic individual was seen in extreme pain, which was originating in the right thigh and leg. There was a pallor of the entire right extremity with absent pulsations of the right dorsalis pedis artery. Pulsation was present only high in the inguinal region. Pulsation of the dorsalis pedis artery and the femoral artery could be easily palpated on the left side. The impression at the time was that she had an embolism to the right iliac or common femoral artery.

Past history revealed that this patient had been treated off and on for mitral stenosis at the Ancker Hospital since 1938. Episodes of dyspnea suggested the presence of heart failure, which was controlled during that period of time with digitalis and quinidine. Auricular fibrillation was present at all times. There was a history of chorea at the age of 14, and she stated that since that time she had had trouble with her heart. The impression at the time she was first seen in 1938 was mitral stenosis of rheumatic origin. An electrocardiogram taken at that time showed an abnormal curve with auricular fibrillation.

Her most recent admission to the outpatient department was for the purpose of investigating bleeding of undetermined etiology from the gastrointestinal tract. Barium enema and an upper gastrointestinal series revealed no evidence of any pathology. It was during a barium enema examination that this last episode occurred. A blood pressure at the time of this most recent admission was 160/80. Pulse was irregular, indicating a presence of auricular fibrillation. It was felt that since the most probable diagnosis was an embolus to the common femoral or iliac artery, early surgery was indicated. Iliac artery embolus was removed through an opening in the common femoral artery by one of us (I. D. B.). Though pulsations were not immediately palpable following surgery, these gradually returned after a period of time with the aid of sympathetic blocks.

Duing convalescence, she had an episode of weakness of the right side of the face and a paresis of the left arm. This came about five days after surgery. It was thought that the cause of this episode was another embolus thrown to the cerebral area.

*When last seen on Feb. 27, 1950, the patient was in excellent spirits.

After this episode, convalescence was smooth until Nov. 26, 1949. At that time, she had a sudden onset of pain in the left calf. The left foot and calf were numb, cold, and pallid. The pulsation which had been previously felt on the left side was now absent. A diagnosis of embolus to the lower extremity on the left side was made. The embolus was removed from the junction of the superficial and deep femoral artery.* It was a successful removal, with pulsation returning immediately.

In summary, therefore, we have a patient with a history of rheumatic mitral disease with at least three episodes of embolic phenomenon. Two of the emboli were removed successfully from both extremities. It was felt, therefore, that this patient would be an excellent candidate for left auricular appendage ligation.

On Dec. 15, 1949, left auricular appendage ligation was done. The patient tolerated the procedure and recovered very nicely. The exact cause of the gastrointestinal bleeding was quite puzzling. Two barium enemas, two upper gastrointestinal studies, and a small bowel study revealed no pathology. The patient remained anemic on the surgical service, but responded well to periodical transfusions.†

Comment.—Various studies[5-8] have suggested that in rheumatic valvular disease the location of intracardiac thrombi is predominantly within the auricles. In some series, the right auricle as well as the left auricle is involved. There appeared to be an even higher incidence of auricular appendage thrombosis in those cases of right auricle involvement than in those of left. It is a well-known fact that pulmonary infarction is a common complication of rheumatic valvular disease.[9] Perhaps a suggestion is in order that symptoms of pulmonary infarction associated with rheumatic heart disease should be an indication for ligation of the right auricular appendage. Garvin[7] has presented evidence that pulmonary infarcts occur much more frequently in the presence of mural thrombi in the right side of the heart than in their absence.

It would be rather presumptious at this time to list the indications and contraindications for ligation of an auricular appendage. Further experience with the procedure should help in the evaluation. Certainly from a review of the literature, the absence of auricular fibrillation is not a contraindication, for embolization can occur in a purely mitral rheumatic heart without this disorder. Burdette has suggested that removal of the appendage may improve the rhythm of the heart. Whether or not it will can be ascertained only by a continued observation of the patients on whom this procedure has been performed. To date none of the patients in our series have stopped fibrillating.

Concerning cerebral embolization, Weiss and Davis[9] have found in a series of forty-eight cases of cerebral embolism that rheumatic heart disease was the probable etiology in twenty-three. This most serious of all complications should lead to a more radical attack on the origin of the embolus. Anticoagulants in cases of cerebral embolus may be of some use, but sometimes may prove to be a factor in the causation of death by intracranial hemorrhage.

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*By Dr. Frank Quattlebaum.

†Since this paper was submitted, this patient was transferred to the medical service for further investigation of the anemia. While on the service, not enough vigorous treatment of the anemia was employed, and the patient expired about two months following original surgery. A post-mortem examination was obtained and no cause for the gastrointestinal bleeding was found. Examination of the heart revealed: (1) a thickened mitral valve; (2) a left auricular appendage which had been previously surgically ligated, with the ostia into the left atrium measuring about 3 or 4 mm. in diameter; and (3) an ante-mortem thrombus within the appendage.

In regard to the technique of ligation, in our first case, a horseshoe type of suture was first placed around the base of the auricular appendage. A secondary tie of umbilical tape was then placed around the base. In the succeeding cases, only a tie of umbilical tape was placed. No effort was made to remove a thrombus if present. We believe that in the future, it would be much better to place a tie of umbilical tape around the base of the auricle, and then to open the tip to remove any thrombus present. A second tie would then be used to close the opening.

SUMMARY AND CONCLUSIONS

1. Three cases in which left auricular appendage ligation has been performed are presented. These are instances of recurrent embolization as a complication of rheumatic mitral stenosis. In each procedure, the course of the patient during the surgical procedure and the immediate postoperative convalescent period was uneventful.

2. It is suggested that ligature of the auricular appendage be used as an additional surgical approach to the problem of recurrent embolization in rheumatic mitral disease. This suggestion is made because of its simplicity as compared to resection of the auricular appendage.

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FUNCTIONAL BRONCHIAL STENOSIS

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THE importance of bronchial stenosis in the development of lung disease has been repeatedly pointed out in publications of recent years. Different authors have described the anatomic causes which produce it and have also pointed out the circumstances under which a permanent stenosis by mechanical transformation may become a transitory valvular stenosis.

The causes of this bronchial stenosis were classified by Leo Eloesser as follows:

- Extrabronchial causes:
  - External compressions
  - Ganglions
  - Aneurysms
  - Hydatid cysts
  - Tumors
- Parietal causes:
  - Tumors
    - benign
    - malignant
  - Bronchiolitis
  - Retractile ulcers
  - Encysted foreign bodies
- Canalicular causes:
  - Loose or free foreign bodies
  - Secretions
  - Mucous plug

These various anatomic causes, however, have not been sufficient to explain the acute and transitory (temporary) syndromes of bronchial stenosis, either because the organic stenosis was not sufficient to justify their existence, or because the acute syndrome has appeared within the chronic evolutionary stage of a bronchial stenosis.

Besides, the clinical observations of partial stenosis which suddenly turn into a reversible or irreversible complete obstruction of the bronchus have led to research work tending to prove the existence of other factors which may cause it.

The bronchographic method has afforded means of studying this stenotic syndrome because the introduction of iodized oil is a procedure which least alters the normal physiology of the bronchus. The first observations of endoscopy and bronchography showed that there exists a normal physiologic cause for the stenosis which occurs during the moment of expiration. It is evident by radioscopy made while the opaque medium is being introduced into the bronchus, that the diameter of the bronchus increases during inspiration and diminishes during expiration. In forced inspiration and expiration this variation in the diameter of the main bronchi is several millimeters. This normal

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physiologic difference of the bronchial caliber is greater with individual variations in children than in adults. The reduction of the bronchial caliber during expiration is accentuated at the origins of the main bronchi, and especially in the lobular trunks where an annular closing of the sphincter type may occur. We have designated these "radicular sphincters." However, it is during cough

Fig. 1.—Bronchography during inspiration and expiration. Note the reduction of caliber of the bronchi. (Figs. 1 to 13 after Di Rienzo: *The Bronchus,* Charles C Thomas, Publisher, 1949.)

Fig. 2.—Bronchograms of the left lung obtained in inspiration and expiration.

that the maximum reduction of the bronchial caliber occurs, and a stenosis takes place which hinders the air from getting out, causing an accentuated retrograde hypertension. During cough, the reduction of the bronchial caliber is not regular, as substantial changes in the parallelism of the walls may be observed, as well as a stenosis which becomes almost a complete obstruction at the sites of the radicular sphincters.

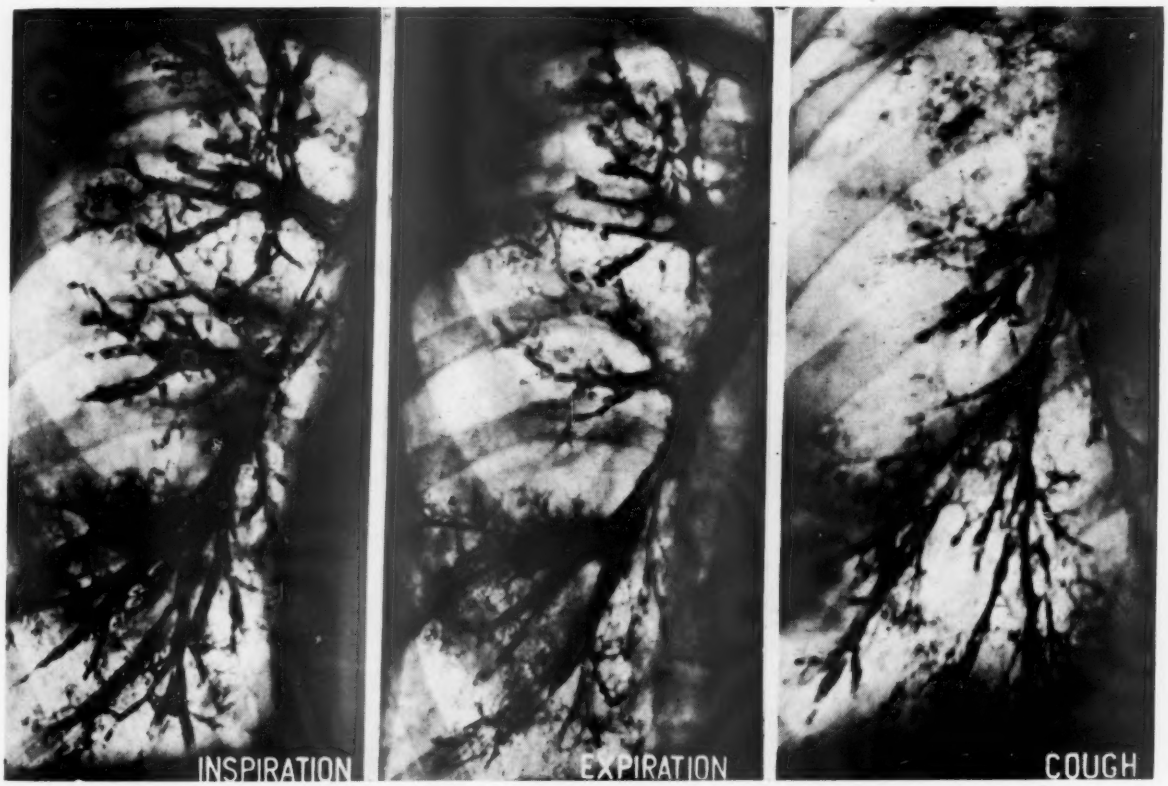

Fig. 3.—Bronchograms taken during inspiration, expiration, and coughing. Note the marked changes in the shape and caliber of the bronchial rami.

The severe bronchotracheal stenosis causes the air column to acquire extraordinary velocity. Assuming that in the act of coughing, which lasts 0.2 to 0.5 second, approximately a liter of air is expulsed, and that this air must pass through tubes which in some places reduce their caliber to 2 or 3 mm. in diameter, some idea will be obtained of the velocity of the expulsed air. Although this velocity has not been measured, it may be assumed to be over 500 M. per second, very nearly that of a pistol bullet. The act of coughing is, therefore, one of the most important factors causing functional bronchial stenosis. Owing to the great intensity of the force of the cough, retrograde hypertension is produced, which overcomes the resistance of the alveoli, causing them to rupture. The undulating contractions in the main bronchi and even in the trachea cause the retrograde hypertension to be greater in some segments than in others. Based upon our dynamic conception of the cough, and the evidence that led to

Fig. 4.—Bronchograms obtained in inspiration and during the act of coughing. Note the undulation of the right bronchus.

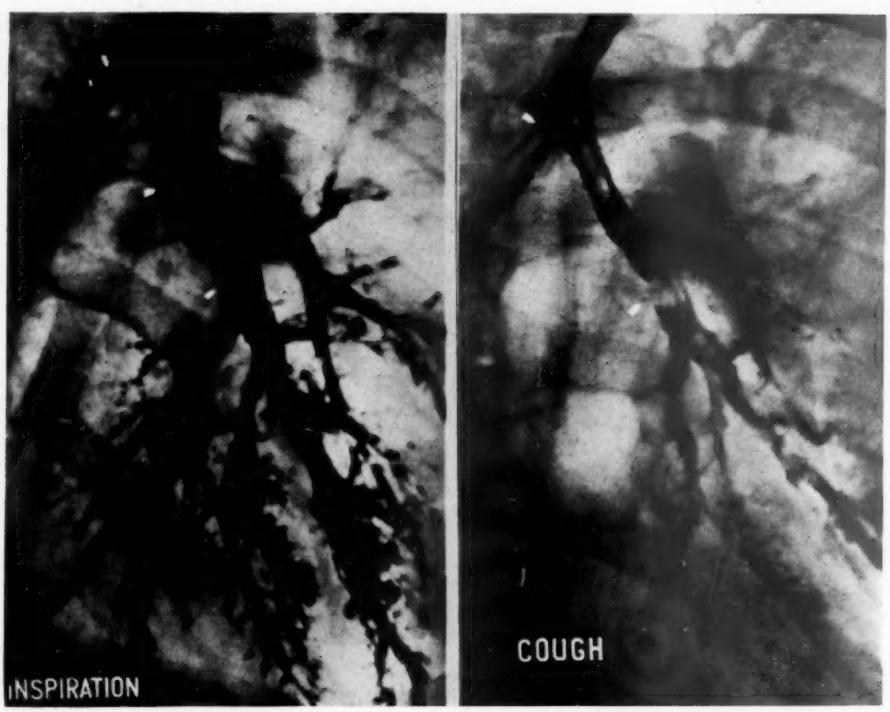

Fig. 5.—Bronchograms obtained during inspiration and coughing. Note the stenosis of the right main bronchus adjacent to the carina.

this concept, we attribute to the cough a primal importance in the genesis of either primary or secondary alveolar and bronchial ectasia. The fact that animals which are unable to cough do not suffer from bronchial ectasia is corroborative evidence of the primary importance of the cough in the pathogenesis of these processes.

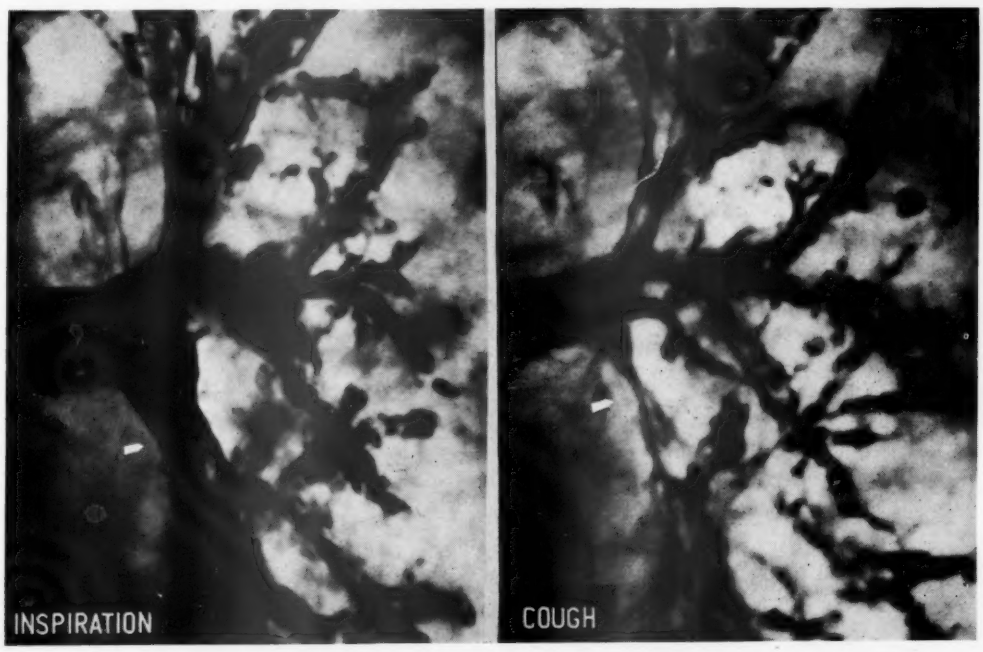

Fig. 6.—Stenosis during coughing at the origin of the left inferior bronchus.

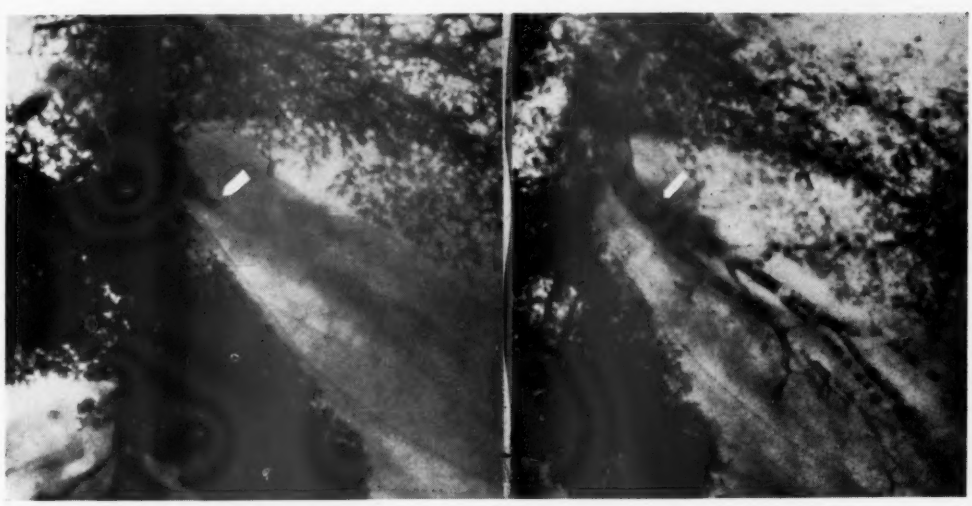

Fig. 7.—Transient stenosis of the principal middle lobe bronchus which disappears following anesthetization of the site of the stenosis.

An example of the dynamic bronchial stenosis is the crying of a child. In some children, the act of crying, with prolonged shouting, causes progressive reduction in the caliber of the main bronchi, with a marked accentuation of

Fig. 8.—Permanent bronchial stenosis at the point of origin of the middle lobe bronchus.

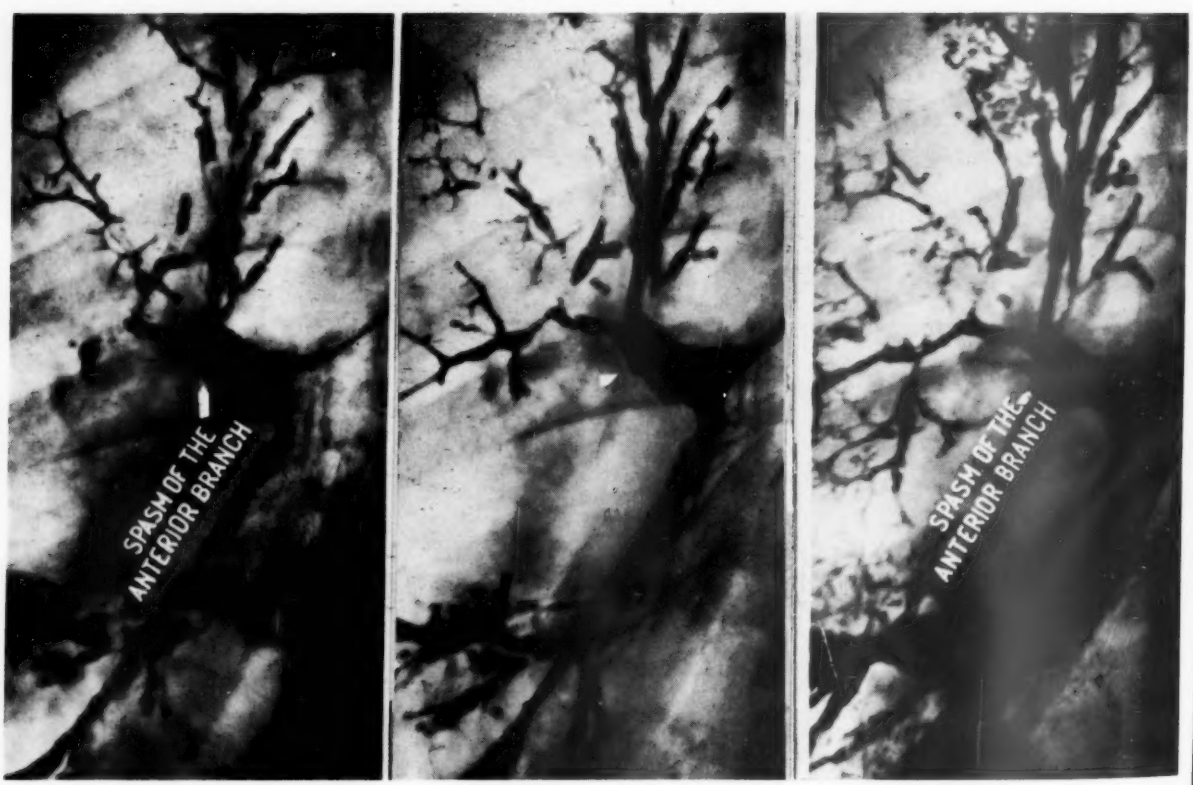

Fig. 9.—Spasm of the bronchial rami during an asthmatic crisis.

stenosis in the radicular sphincterial system. This prolonged crying is likely to precipitate a stenosis which may lead to an acute paroxysmal asphyxia.

Besides this dynamic functional stenosis of a physiologic character which may be accentuated by pathologic factors, there are others static, permanent, or transitory, which are exclusively pathologic. The transitory static func-

Fig. 10.—Spasm of the right main bronchus and its rami during an asthmatic crisis.

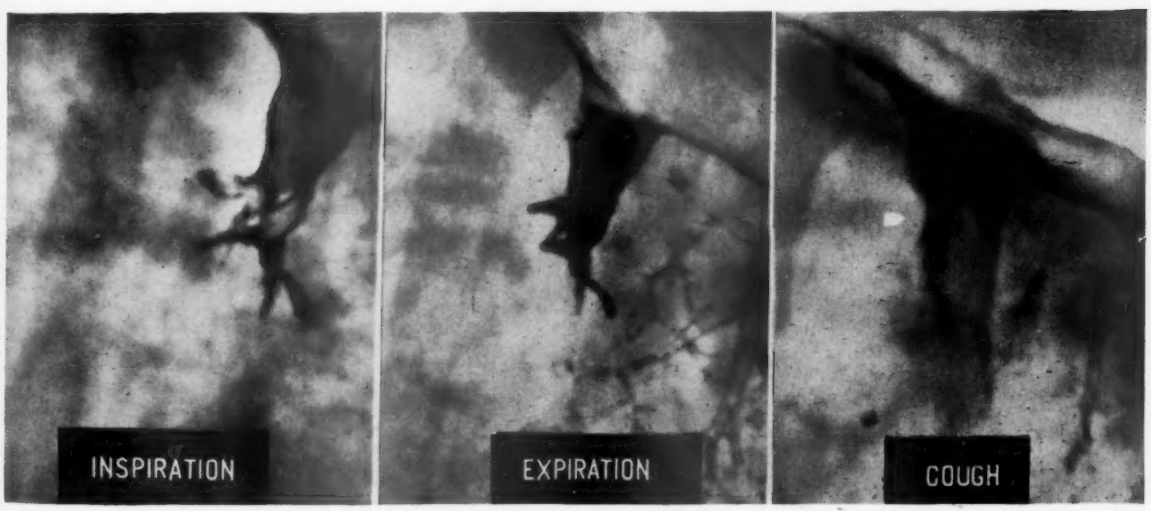

Fig. 11.—Bronchograms of inspiration, expiration, and coughing during an asthmatic crisis.

tional stenosis may be observed in an asthmatic crisis during attacks of bronchitis, and as a reflex phenomenon following an abdominal operation. This stenosis may progress to complete obstruction of the bronchial lumen and produce segmentary or lobular atelectasis. Laminary and transient atelectasis and those types without other manifest etiology are probably caused by these

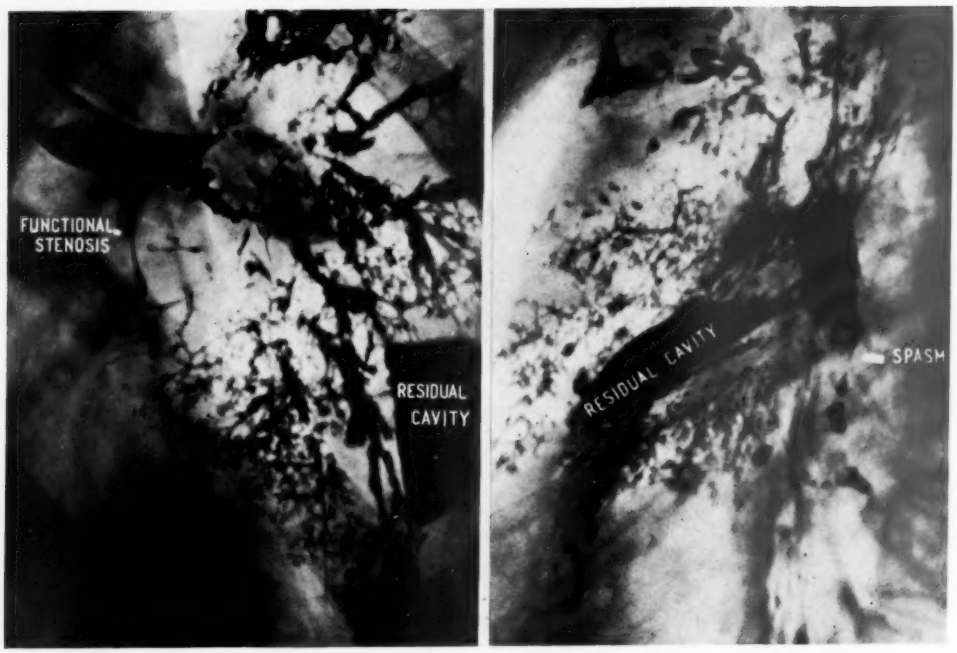

Fig. 12.—Functional bronchial stenosis appearing postoperatively in the left inferior bronchus following removal of a hydatid cyst.

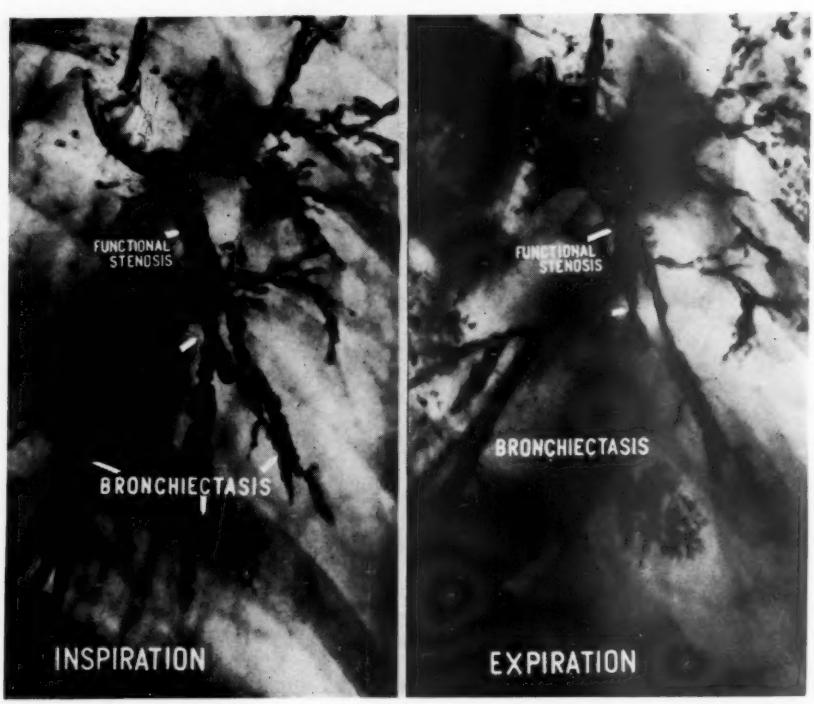

Fig. 13.—The same patient after anesthetizing the stenosed bronchus.

functional obstructions. There are also permanent static functional stenoses as observed in some patients with bronchiectases, or in others after measles and whooping cough.

These transitory or permanent functional stenoses can be demonstrated by the bronchographic method, and are attenuated or may disappear with anesthesia when vasoconstrictor substances, such as Adrenalin or Privine, are added to the anesthesia. These stenoses always occur at the root of the branches, especially of the lobular trunks. The transitory or permanent functional stenoses, static or dynamic, have not only been suspected by their clinical characteristics, but they have been unquestionably proved by the bronchographic method.

It is apparent that not only the muscular fibers, but also other elastic properties of the mucous membrane and the vascular system participate in the genesis of bronchiectasis. This is evident because in order to relieve the stenosis it is absolutely necessary to add a vasoconstrictor substance to the anesthetic agent. Moreover, when the stenosis is accompanied by mucous membrane plication, both disappear simultaneously. The stenosis is caused by the contraction of muscular fibers and the erectile property of the mucous membrane. The muscular fibers alone are not anatomically able to cause a complete obstruction of the bronchus, but their contraction together with that of the mucous membrane produce the functional obstruction which is clinically observed. This may originate processes which eventuate in organic strictures.

Mazzei has magnificently described the classical pictures initiated by bronchial stenosis. It is for this reason that we believe that the classification of bronchial stenosis as made by Leo Eloesser should be completed by adding the following functional types:

Dynamic:
- Expiration
- Crying
- Coughing

Static:
- Transitory
- Permanent

It would still be necessary to add those types of composite etiology in which the organic factors are combined with the functional (as observed, for instance, in cases with an intrabronchial foreign body), but which are more frequently present in most lesions which begin with an infection of the bronchial mucous membrane.

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THE RUPTURED APPENDIX

A Survey of Five Hundred and Forty-One Cases, 1939 Through 1948

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INTRODUCTION

"SUPPURATION upon a protracted pain of the parts about the bowels is bad." Hippocrates.

It is recognized that we of the medical profession are living in a golden age of medical therapeutics in which one important discovery is soon supplanted by an even more important adjunct of therapy. The data to be presented are the findings of a ten-year survey of the experiences with ruptured appendix at the John Gaston Hospital, Memphis, Tenn., the teaching hospital of the University of Tennessee College of Medicine. During this period of time the fundamental surgical philosophy in the management of these cases has remained practically unaltered. It is with the advent of chemotherapy and antibiotics and the freer use of blood and blood derivatives that noteworthy improvement in results has been made. The proposed problem is to show to what extent these therapeutic advances have enabled us to lower the mortality rate for the ruptured appendix in our hospital.

HISTORICAL ASPECTS

The first reference to the appendix in recorded literature was in 1522 A.D., when Berengarius Carpus, professor of surgery at Pavia and Bologna, wrote of an *addi tomentum* at the end of the cecum. Following this original description, various writers, including Vesalius, Stephanus, Ambroise Parè, Fallopian and Laurentius, discussed the appendix in their writings. The gross anatomy of the appendix has been well known since the close of the eighteenth century. However, the microscopic anatomy was not disclosed until much later, some of the best studies being those of Lockwood.[8]

Hippocrates made the first recorded observation that "Suppuration upon a protracted pain of the parts about the bowels is bad." And in another statement, Hippocrates wrote, "From the rupture of an internal abscess, prostration of strength, vomiting and death ensue."

The earliest recorded medical case of perforated appendicitis, followed by death and necropsy, was by Fernelius, in 1567. The term appendicitis was not used, but it was described as an "affection of the cecum." Heister, in 1753, published a case, with autopsy, describing a gangrenous appendix lying in a collection of pus, and proving, said Heister, that "This part can be the seat of inflammation and abscess." Later, in 1815, the first American case of ruptured appendicitis was recorded by Prescott and the case authenticated by necropsy.

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†Deceased.

In 1824, Lauyer-Willermay described two cases of peritonitis due to perforated appendicitis. Von Merling, in 1836, published a monograph on the pathologic anatomy of the vermiform appendix.

James Copland, in 1834, was the first to discriminate between inflammations of the cecum, the vermiform appendix, and the pericecal tissue. And Hodgkin, in 1836, recognized that the appendix was responsible for both local abscesses of the right iliac fossa and of diffuse peritonitis. Burne, in 1837, published a concept maintaining that diseases of the right iliac fossa originated in the cecum; however, two years later he reversed his viewpoint and stated that right iliac disease was caused more commonly by a primary process in the appendix. Burne's latter viewpoint was substantiated by Valz, in 1843, and later by Lierdet, in 1859, both of whom maintained that the appendix was responsible for the majority of the disease processes in the right lower quadrant.

Rokitansky, in 1841, accurately described diseases of the vermiform appendix in the following words: "Catarrhal inflammation of the vermicular process is a disease of common occurrence, and is very dangerous on account of its consequences. It much resembles typhlitis stercoralis and is invariably the result of fecal matters and foreign bodies, especially small fruit stones having become lodged and hardened in it."

Fitz,[5] in 1886, published his epochal article which convinced the medical world of that time that disease of the vermiform appendix was the most common cause of all inflammations of the right iliac fossa. He collected 257 cases of inflammation of the perforated appendix, and 209 cases of so-called typhlitis and perityphlitis, and, by showing that the symptoms of the two conditions were the same, founded the convincing truth that they were etiologically identical. The term appendicitis was first used by Fitz in his excellent paper. He also pointed out that the resulting cases of generalized peritonitis in the majority of instances appear on the second, third, and fourth days after the inflammation of the appendix is established. Death ensues as a result of the peritonitis, in the first week in 56 per cent, in the second week in 31 per cent, and in the third week in 4 per cent. Furthermore, 68 per cent of patients with fatal cases die within the first eight days of the illness, and two-thirds of these die between the fourth and eighth days.

TREATMENT OF THE RUPTURED APPENDIX

The treatment of appendicitis varied through the years, and very few cases are found in the literature of surgery before 1880 to 1900, when there was a tendency toward the surgical rather than the medical treatment of appendicitis. This surgical wave seemed to sweep over the entire country. In 1883, Pepper[11] stated that, "The unjustifiable delay permitted in many cases of typhlitis whilst hoping day after day for the more definite detection of suppuration was the direct cause of many avoidable deaths."

However, opinions were divided. In 1892, Young[14] pleaded with physicians for less surgical interference in the management of appendicitis, and advocated a medical regime of hot fomentations, enemas, and morphine. He

stated that the errors in diagnosis and the numerous and grave dangers of an operation did not warrant a laparotomy.

A combined medical and surgical regime was also used. In 1886, Fitz wrote that keeping the bowels quiet should be the first and last thought, and that a laxative or cathartic might be the means of exciting a generalized peritonitis. He also advocated absolute rest in bed, liquid diet in small quantities often repeated, and above all, sufficient opium to neutralize pain. Then, if after the first twenty-four hours from the onset of severe pain the peritonitis was evidently spreading, an immediate operation for exposing the appendix and determining its condition with reference to its removal was indicated.

There were actually three schools of thought in the treatment of appendicitis: the first, to treat entirely conservatively; the second, to operate as soon after rupture as possible if localization was not taking place; and the third, not to operate until abscess formation had occurred. The mortality rate was extremely high in all three categories.

As years passed, in 1910 and later, the trend toward a more aggressive attitude in the surgical treatment of acute appendicitis—and especially so toward ruptured appendix—was more pronounced. Chideckel[3] and Baruch[1] were pioneers in this bold venture, the latter coining the phrase, "When in doubt operate." Schullinger[12] reported a mortality rate in the years 1918 to 1920 of as high as 30 per cent in patients with generalized peritonitis, and as high as 20 per cent in localized abscesses.

Later in the 1920's and early in the 1930's operation was advocated universally, in acute appendicitis and also in early perforation. Operation in the face of diffuse peritonitis was abandoned, and most physicians followed the treatment of Ochsner, which advocated stomach tube washings, bed rest, morphine, Fowler's position, and so forth, when peritonitis was present. Schullinger reported a mortality rate in this period of about 20 to 35 per cent in patients with generalized peritonitis and about 6 to 12 per cent in those with localized abscess. Boland[2] of Atlanta, in 1932, in an analysis of all hospitals, found 33 per cent mortality in generalized peritonitis.

In 1933, Wangensteen[13] introduced the continuous suction siphonage apparatus attached to the Levine gastroduodenal catheter. At about the same time the importance of replacing the fluids and electrolytes lost by vomiting and suction was generally recognized, due to observations of many investigators. Among the most important contributors were Hoguet,[7] Gamble,[6] and many others. The use of oxygen inhalations, as advocated by Fine,[4] was introduced and gradually the mortality rate in patients with ruptured appendices was lowered. There was some improvement in statistical reports, but there was no indication that the future held great promise despite these historically important adjuncts. Suction siphonage, fluid and electrolyte replacements, and oxygen were indeed responsible in adding support to a failing organism, but the last word was still to be written.

THE PRESENT SERIES

The John Gaston Hospital is the free hospital of the city of Memphis. More than 90 per cent of the general surgical patients are operated upon by

the resident and interne staff, while in patients with appendicitis this percentage approaches 100 per cent. The following review, therefore, represents the attending staff-supervised results of the resident surgical group. All surgical patients are seen daily by one or more attending staff surgeons accompanied by the resident surgical group. It should be recorded also that at least 80 per cent of the patients are Negro. The average red blood cell count hovers about the 3,000,000 mark and the hemoglobin percentage is approximately 60 as found in the review of these two important factors in this hospital.

TABLE I. MORTALITY STATISTICS (1929 TO 1939)*

YEAR	PATIENTS	RECOVERED	DEATHS	MORTALITY (%)
1929	19	12	7	42.10
1930	32	27	5	15.62
1931	13	9	4	30.80
1932	39	24	15	38.46
1933	38	26	12	31.57
1934	53	32	21	39.62
1935	46	27	19	41.30
1936	66	40	26	39.43
1937	68	52	16	23.53
1938	68	59	9	13.23
Total	442	308	134	

*Ten-year over-all mortality rate, 30.3 per cent.

In an effort to review our results in the past decade in the treatment of the ruptured appendix, the records of the previous decade, 1929 through 1938, were compiled as shown in Table I. It reveals a dismal picture, no worse nor better, however, than the average charity hospital series of comparable years. There were 442 cases of ruptured appendices, with 308 recoveries and 134 deaths, and a ten-year over-all mortality rate of 30.3 per cent. In 1932, Boland's over-all review revealed a 33 per cent mortality rate. It should be recalled that since 1934, suction siphonage, fluid replacements, and oxygen were adjuncts freely employed and judiciously administered. There was no perceptible statistical improvement excepting in the years 1930 (15.62 per cent) and in 1938 (13.23 per cent).

The past decade, with the free employment of the sulfonamides during the earlier years, and the antibiotics penicillin and streptomycin in the latter years, along with all our former adjuncts presents a brighter picture.

Duration of Symptoms.—Despite the publicity campaigns of our generation, the lay public, and, alas, many physicians, are responsible for much delay

TABLE II. PURGATION BEFORE ADMISSION

YEAR	PREADMISSION PURGATION	PERCENTAGE	MORTALITY PERCENTAGE
1939	37 in 71 cases	52.1	14.08
1940	40 in 63 cases	63.5	12.69
1941	32 in 69 cases	49.3	4.34
1942	39 in 64 cases	60.9	20.00
1943	20 in 32 cases	60.6	21.21
1944	13 in 24 cases	54.1	12.50
1945	23 in 40 cases	57.5	17.50
1946	33 in 80 cases	41.2	11.25
1947	19 in 40 cases	47.5	2.50
1948	40 in 56 cases	71.4	0.00

and too much purgation. Fig. 1 shows that only 77 patients in the series of 541 were brought to the hospital in the first day of the disease. Over 60 per cent were admitted after forty-eight hours.

Purgation Before Admission.—The most embarrassing picture is detailed in Table II. Purgatives had been given one or more times prior to admission in 1948 in 71.4 per cent of the patients admitted in this enlightened year. This series of preadmission purgations was investigated in order to ascertain a relationship between purgation and the mortality rate. No startling conclusions can be drawn, however.

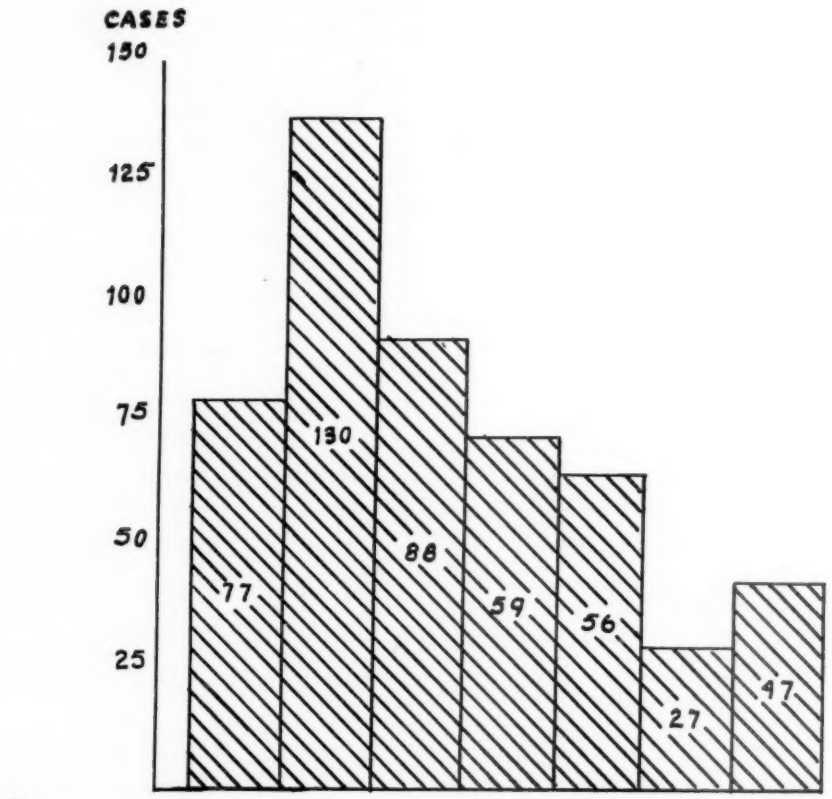

Fig. 1.—Duration of symptoms before admission.

Admission Temperatures.—As in most series reported, the admission temperatures range between + 99° and + 103° F., with the majority of patients' temperatures ranging between + 100° and + 102° F.

Admission Pulse Rates.—The majority of the pulse rates, as in comparative series, ranged between 80 and 120 per minute. Admission pulse rates above 120 revealed a much poorer prognosis and the majority of the deaths were in this group.

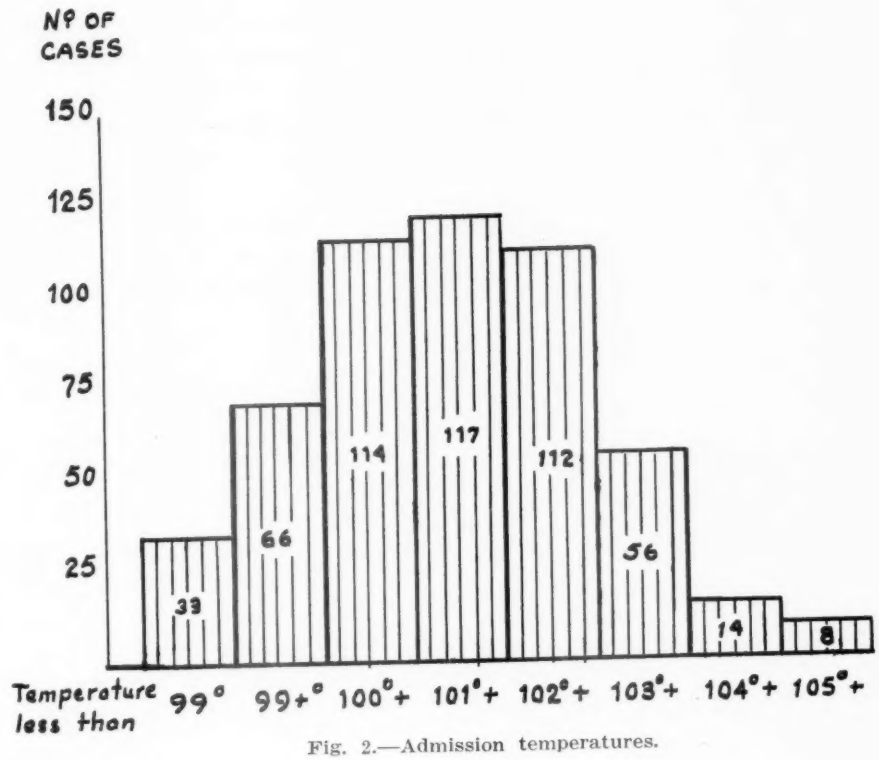

Fig. 2.—Admission temperatures.

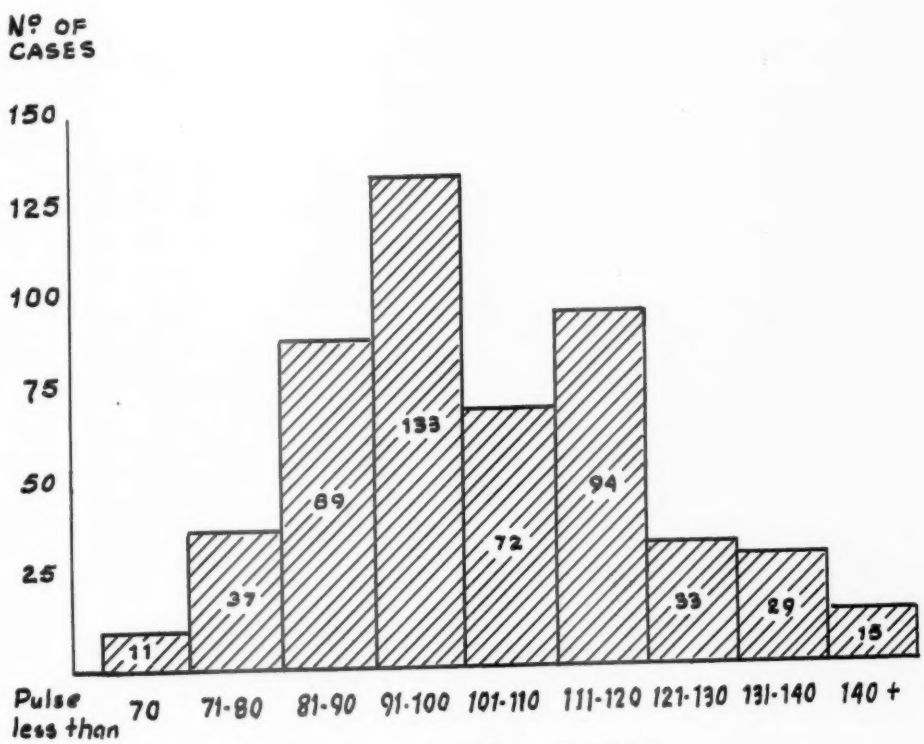

Fig. 3.—Admission pulse rates.

The Leucocytic Response.—The leucocytic response was recorded on admission in 493 patients out of the total of 541 in the series. Totals of more than 12,000 were revealed in 68.9 per cent of the admission counts. Of the seventy-four patients with counts below 8,000, there were thirty-one deaths, just over

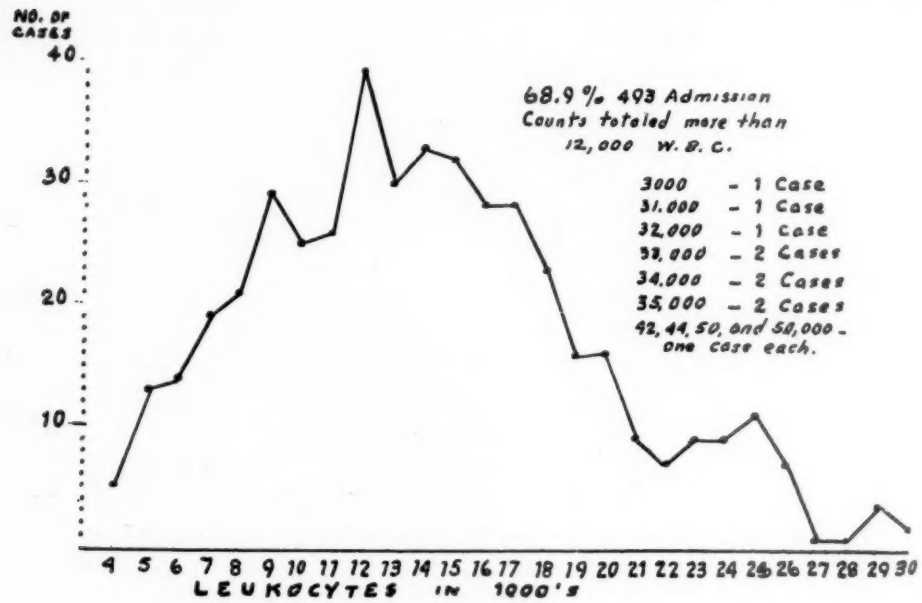

Fig. 4.—Admission total leucocyte counts.

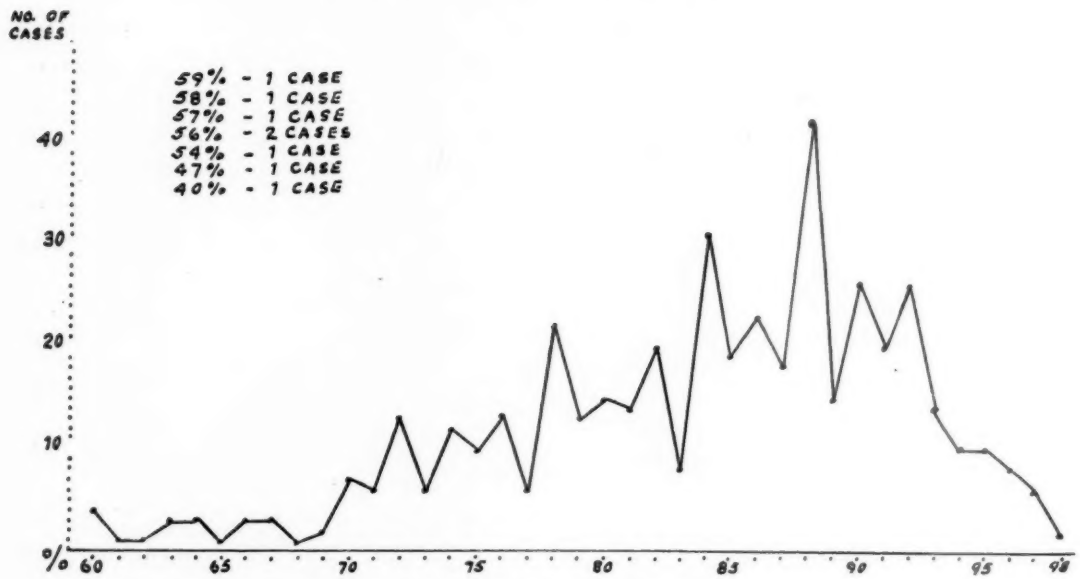

Fig. 5.—Admission polymorphonuclear counts.

the 50 per cent mark of the total deaths in the entire series. There was a good shift to the left in the differential leucocyte counts, as will be noted in Fig. 5, the majority of the 493 recorded differential counts ranging well over 80 per cent, including juvenile and stab forms.

TABLE III. ACUTE DIFFUSE AND GANGRENOUS APPENDICITIS
(1939 TO 1948 INCLUSIVE)

YEAR	ACUTE DIFFUSE APPENDICITIS*		ACUTE GANGRENOUS APPENDICITIS†	
	PATIENTS	DEATHS	PATIENTS	DEATHS
1939	253	2	2	0
1940	369	0	23	1
1941	477	0	18	1
1942	416	0	15	1
1943	230	1	16	1
1944	206	2	17	0
1945	208	0	16	0
1946	257	0	16	1
1947	115	0	21	0
1948	114	0	31	0
Totals	2645	5	175	5

*Mortality rate, 00.18 per cent.
†Mortality rate, 2.85 per cent.

Mortality Statistics.—During the period under observation, 1939 to 1948 inclusive, there were 3,396 cases of acute appendicitis of all grades and types with an over-all mortality of seventy-one. The mortality percentage in this combined grouping was 2.09 per cent.

In Table III are recorded in separate series acute diffuse appendicitis and gangrenous appendicitis according to the reports of the department of pathology. There were 2,680 cases of acute diffuse appendicitis with five deaths, a mortality rate of 0.18 per cent—less than ⅕ of 1 per cent. One of these patients died of a blown-out stump, the only death from peritonitis. Only five deaths were recorded from acute gangrenous appendicitis in a series of 175 cases, a mortality rate of only 2.85 per cent. This is a remarkable improvement in mortality in such a large series. Two of the deaths were due to pylephlebitis.

The Ruptured Appendix.—In the 541 cases of ruptured appendix there were sixty-one deaths. Of the total number of 3,396 patients admitted in the ten-year period, 541 or 15.9 per cent were admitted to the hospital with a ruptured appendix and peritonitis. The fact could be interpreted that approximately every sixth patient admitted with a diagnosis of appendicitis had peritonitis. This fact is a sad commentary on the results of our efforts in education at professional as well as at public levels.

TABLE IV. THE RUPTURED APPENDIX SERIES, MORTALITY STATISTICS

YEAR	PATIENTS	RECOVERED	DEATHS	MORTALITY (%)
1939	71	61	10	14.08
1940	63	55	8	12.69
1941	69	66	3	4.34
1942	65	52	13	20.00
1943	33	26	7	21.21
1944	24	21	3	12.50
1945	40	33	7	17.50
1946	80	71	9	11.25
1947	40	39	1	2.50
1948	56	56	0	0.00
Totals	541	480	61	11 27

The best results are recorded in the years 1947 and 1948 (see Table XII), when the antibiotics, penicillin and streptomycin, were used in all cases along with intravenous sulfadiazine.

There is one interesting observation that should be noted in the prewar years, 1936 to 1942. Though the antibiotics were practically unknown during that period, and the sulfonamides used fully only in the last of that span of years (1941), a dramatic drop in the death rate occurred. The surgical staff, both attending and resident, were well organized in the planned over-all attack upon appendical peritonitis. The confusion and chaos of the following four war years, 1942 to 1946, found reflection in the increased mortality which accompanied this span of years. Most of the active attending staff was in the service and the greatly overworked and abbreviated "9-9-9" resident staff did the best it was capable of under the most harassing conditions and shortages of men, materials, and drugs. By 1941 the mortality rate had been lowered to the surprising figure of 4.34 per cent, a steady downward curve from 41.3 per cent in 1935 (the first full year of suction-siphonage and fluid replacement therapy). It is our consensus that had not World War II interrupted the well-established teamwork of the attending and resident groups, the ten-year rate would have been closer to 5 per cent than the 11.27 per cent. This is mere conjecture, but the figures were definitely toward that trend at the end of 1941.

Decade Mortality Rate.—The decade mortality rate reveals the highest mortality rates in the sixth and seventh decades (50 to 70 years). In the eighth decade, with only one death in eight patients, the rate was but 12.5 per cent. There were no deaths in two patients in the ninth decade. Of the men or women who survive the dangerous years, 50 to 70, nature has endowed those past 70 with vascular systems which withstand much. Our observations are similar in acute cholecystitis and in ruptured peptic ulcers. In a group of seventy-seven patients over 50 years old there were fifteen deaths, a mortality rate of only 19.3 per cent.

TABLE V. MORTALITY STATISTICS, DECADE MORTALITY RATE

DECADE	AGE (YRS.)	NUMBER OF PATIENTS	DEATHS	MORTALITY (%)
1	(0 to 10)	87	8	9.2
2	(11 to 20)	139	11	7.9
3	(21 to 30)	87	8	9.2
4	(31 to 40)	89	12	13.6
5	(41 to 50)	62	7	11.3
6	(51 to 60)	44	9	20.4
7	(61 to 70)	23	5	21.7
8	(71 to 80)	8	1	12.5
9	(81 to 90)	2	0	0.0
	Totals	541	61	

By far the greater number of ruptured appendices occurred in the second decade. Here, in this same decade, is found the lowest mortality rate—7.9 per cent of 139 patients. The mortality of 9.2 per cent in children 10 years of age or under compares well with Norris' report[9] of 10.76 per cent mortality

from the ruptured appendices in a review of 1,000 cases in children with appendicitis of all degrees. His over-all mortality in the 1,000 cases was 3.2 per cent.

Race Mortality Rates.—In comparing the mortality rates in the white and Negro groups, where there were four Negro patients to one white, there were significant differences. There were marked disparities between male and female rates in both group. The Negro male mortality was 10.8 per cent, while that of the white was but 6.78 per cent. The Negro female rate was the greatest of all four, reaching 16 per cent in comparison to 12 per cent in the white female group. It may be inferred that the female groups presented more difficult diagnostic problems before hospital admission. On reflection it seems most significant that the female patients, as a rule, seemed in a distinctly more critical condition upon admission than did the males, and their hospital courses seemed stormier. The physical and economic levels of both white and Negro are factors that may bear serious weight in the disparities also. The Negro standards in both these levels were, broadly, lower than the white.

TABLE VI. COMPARATIVE MORTALITY RATES: WHITE AND NEGRO

	WHITE (9.17%)			NEGRO (11.81%)		
	MALE	FEMALE	TOTAL	MALE	FEMALE	TOTAL
Patients	59	50	109	307	125	432
Death	4	6		31	20	
Mortality (%)	6.78	12.00		10.80	16.00	

Appendical Abscess.—There were 130 appendical abscesses in the entire group, with eight deaths, a mortality rate of 6.15 per cent. This rate compares well with Schullinger's report (6 to 12 per cent). In the latter three years there was but one death (in 1947) among fifty-three patients with appendical abscess. This one death, the only death in the 1947 group, occurred in a 52-year-old Negro man. He died on the fifth day after admission, following a preadmission illness of four days. He was too ill at any time for surgical intervention. Autopsy revealed a tremendous apendical abscess. Of the remaining 411 patients in the entire series, forty-six died, a mortality rate of 11.19 per cent.

TABLE VII. APPENDICAL ABSCESS*

YEAR	PATIENTS	DEATHS
1939	23	2
1940	20	2
1941	13	1
1942	8	0
1943	2	0
1944	3	0
1945	8	2
1946	19	0
1947	17	1
1948	17	0
Totals	130	8

*Mortality rate, 6.15 per cent. Of 411 patients remaining, 46 died, a mortality rate of 11.19 per cent.

Deaths From Time of Admission.—The deaths, from the time of admission, are detailed in Fig. 6. Of thirty-two patients who died in the first week of admission (52.8 per cent of the total deaths), twenty died with no surgery possible, seven on the third day and five on the second day. Two died on the day of admission, one in the receiving ward before admission could be made to the wards. This is a remarkable comparison with Fitz' series in 1886, in which 56 per cent of his 466 patients died in the first week. Of the total number of sixty-one deaths, there were thirty-one within two weeks of admission without surgical intervention. These patients remained persistently critically ill, showing not the slightest improvement to warrant any surgical trauma. In all, thirty-four died without

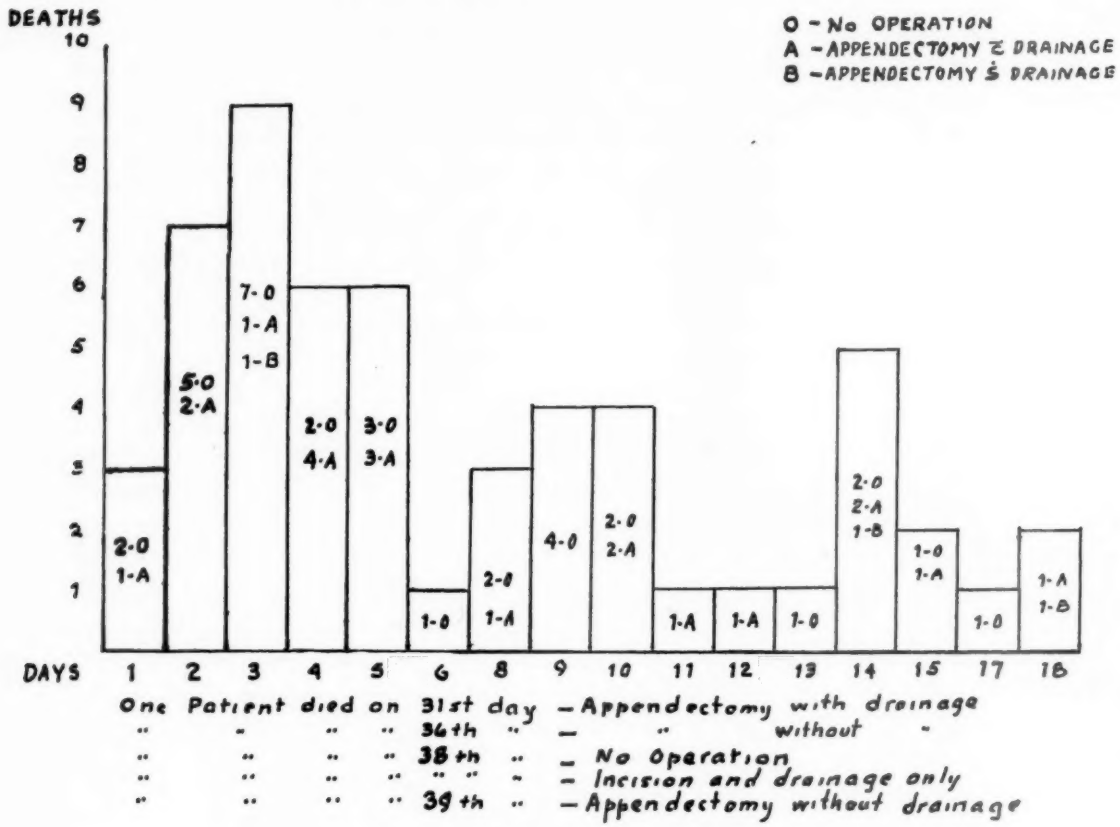

Fig. 6.—Deaths from time of admission.

any surgery. Thus, well over one-half the deaths occurred in patients who remained in irretrievably critical condition from the time of their admission. In no case were only intensive medical and mechanical means employed when surgery might have been tried. In comparison with Fitz' series, in which 31 per cent of the patients died in the second week, there were nineteen deaths. Again, Fitz' figure represented 31 per cent of all patients in his series of 466 cases.

Surgical Procedures.—In Table VIII are detailed the procedures employed in surgical intervention and the mortality in each procedure. The Richardson-

TABLE VIII. SURGERY AND MORTALITY

DEATHS	TREATMENT
34	No operation*
21	Appendectomy with drainage
5	Appendectomy without drainage
1	Incision and drainage
61 Total deaths	

*Twenty patients died within the first week, no surgery; thirty-one patients died within two weeks, no surgery.

McBurney incision was the technique used in all patients operated upon. In the majority (372) operation consisted of appendectomy with drainage. There were twenty-one deaths in this group, a mortality of 5.64 per cent. The controversial appendectomy in ruptured appendices without drainage was done in seventy-seven instances with five deaths, a mortality rate of 6.49 per cent. The following rule is now rigidly enforced in this controversy: If the appendix is ruptured, the disease is macroscopically so well localized that no free fecal contamination has occurred, and the area of involvement is easily seen and thoroughly removed, then appendectomy without drainage may be performed. If there is any question of an escaped fecolith, particularly in basal rupture, drainage should be employed.

It is emphasized here, again, that an irretrievable number of patients, upon whom no surgery can be performed because they are *in extremis,* comprised the majority of the deaths in the over-all series. Of a total of sixty-one deaths, thirty-four patients died before surgery dared be attempted. "Suppuration upon a protracted pain of the parts about the bowels is bad." It was in Hippocrates' time, and remains so.

TABLE IX. TYPES OF OPERATIONS PERFORMED

Immediate appendectomy with drainage	317
Immediate appendectomy without drainage	41
Incision and drainage of abscess only	7
Delayed appendectomy with drainage	65
Delayed appendectomy without drainage	36
No operation	
Discharged, to return for surgery	41
Died	34
Total	541

A summary of the types of operations performed follows in Table IX. It will be noted that only 7 of the 130 appendical abscesses were incised and drained. Intensive medical and mechanical treatment with careful observations aided immeasurably in this group of patients. It was most heartening to follow, almost daily, the recession in abscess. Incision and drainage were employed only when the abscess was noted as stationary or where possible extension or rerupture was diagnosed or suspected. Only one death resulted in the group of operations, which are usually critical procedures in themselves. In the entire series there were seventy-five patients not operated upon. Of these,

forty-one were discharged from the hospital with arrested conditions to return for surgery in ten weeks (an arbitrary minimum limit), and thirty-four died in the hospital before surgery could be employed.

To minimize intra-abdominal and intraperitoneal trauma, the right lower quadrant approach (Richardson-McBurney incision) was used in all cases of surgery. The appendical stump was inverted in the majority of cases. In some, simple amputation after stump ligation was used, and in a few, where the inflammatory process extended beyond the base, the Parker-Kerr type of amputation was employed.

There was no uniformity in the kind or type of drainage employed. Early in the series sulfanilamide crystals were liberally sprinkled about the diseased area and a Penrose drain or two were used. Where large contaminated areas were found the drains were multiplied, and, occasionally, a thin vaginal gauze pack impregnated with sulfanilamide crystals to stimulate exudation was used. Intraperitoneal antibiotics, though not frowned upon, have not found favor. The systemic attack against peritonitis, apparently, is the treatment of choice at the present writing. In the abdominal wall incision we encourage the use of sulfanilamide crystals, not so much for their antibacterial potency, but because they stimulate an exudative and fluid flow toward the open or drained incision. The sulfonamides are excellent local fibrinolytic agents as well, and aid in maintaining free drainage.

TABLE X. COMPLICATIONS

PROVED DIAGNOSIS	NUMBER OF CASES
Pelvic abscess	12
Bronchopneumonia	12
Acute intestinal obstruction	11
Fecal fistula	10
Paralytic ileus	10
Mild wound infection	10
Subphrenic abscess	7
Lobar pneumonia	5
Atelectasis	5
Abdominal wall abscess	4
Severe wound infection	4
Thoracic empyema	3
Lung abscess	3
Toxic hepatitis	3
Pylephlebitis	2
Subhepatic abscess	2
Hepatic abscess	2
Late pregnancy	2
Thrombophlebitis	2
Uremia	2
Incisional hernia	2
Syphilis	2
Strangulated hernia, acute bronchitis, hypertensive cardiovascular disease, retroperitoneal abscess, pulmonary embolus, orchitis, vasitis, measles, chicken pox, blown-out base of amputated appendix, hemorrhage from incision, typhoid fever, pulmonary tuberculosis, tuberculous peritonitis, and sulfonamide dermatitis.	1 each

Complications.—As in any large series where peritonitis, localized or generalized, is studied, numerous complications and some concomitant primary diseases may be noted. In Table X are listed the complications and concomitant diseases in our series. It should be noted that there were no eviscerations nor wound dehiscences. Two incisional hernias were late manifestations. Of the ten fecal fistulas, all closed without further surgery. Of the dreaded residual abscesses, there were twelve pelvic, seven subphrenic and two subhepatic abscesses. Three thoracic empyemas were the result of subphrenic extension. The two cases of pylephlebitis occurred in the patients where gangrenous appendices had ruptured. In the entire series, 120 major and minor complications are listed. It is interesting to note that there was but one therapeutic complication, sulfonamide dermatitis. It should be noted that thousands of doses of sulfadiazine were employed in this series, as will be detailed later.

Causes of Death.—The causes of death were divided into nonoperative and operative series in Table XI. As might be surmised, the majority of the deaths (41) were caused by generalized peritonitis. In addition, there were eight deaths from appendical abscess. It will be noted that there were two deaths from generalized peritonitis in the last trimester of pregnancy. Whether there is present a more virulent peritonitis, or a greatly displaced appendix, high in the right upper quadrant (obviously difficult to diagnose), the problem is difficult to solve. Peritonitis late in pregnancy always carries a prohibitive mortality rate. In almost every series of ruptured appendices there is a case in which a ruptured appendix is found complicating a strangulated inguinal hernia. This series is no exception. Though approximately one-seventh of the series was in the age group past 50 years (77 in 541), no deaths were caused incident to age, or cardiac, nephritic, or vascular conditions.

TABLE XI. CAUSES OF DEATH

AUTOPSY		NO AUTOPSY	
Nonoperative Deaths			
Generalized peritonitis	18	Generalized peritonitis*	10
Appendical abscess	2	Appendical abscess	3
Lung abscess	1		
Totals	21		13
Operative Deaths			
Generalized peritonitis	6	Generalized peritonitis	7
Pulmonary abscess	1	Appendical abscess	3
Typhoid fever	1	Pylephlebitis	2
		Acute intestinal obstruction	2
		Subphrenic abscess	1
		Lung abscess	1
		Lobar pneumonia	1
		Hemorrhage	1
		Strangulated inguinal hernia	1
Totals	8		19
Deaths from generalized peritonitis	41		
Deaths from appendical abscess	8		

*Two died in late pregnancy.

TABLE XII. THE NEW THERAPY

YEAR	SULFONAMIDE		PENICILLIN		SULFONAMIDE AND PENICILLIN		STREPTOMYCIN, PENICILLIN, AND SULFONAMIDE	
	PATIENTS	DEATHS	PATIENTS	DEATHS	PATIENTS	DEATHS	PATIENTS	DEATHS
1939	4	1						
1940	53	6						
1941	60	3						
1942	57	11						
1943	31	6						
1944	19	1						
1945	15	2	5	2	17	2		
1946			10	2	68	7		
1947	2	0	3	1	33	0		
1948*			1	0	27	0	27	0

*One patient, in 1948, received streptomycin and penicillin.

The New Therapy.—This series had its beginning in the year that sulfadiazine for intravenous use was available in liberal quantity, and sulfanilamide crystals and powder were used freely. Penicillin was first made available to our charity hospital late in 1945.

In Table XII are tabulated the drugs, chemical and antibiotic, which were used. It is significant to note that in 1941, when sixty of the sixty-nine patients in that year received sulfadiazine and large doses of sulfanilamide crystals locally and intraperitoneally, there were but three deaths. Then the war years intervened. In 1945 penicillin was used in approximately one-half of the patients. However, in 1946 seventy-eight of the eighty patients received penicillin and the sulfonamide drugs. Intravenous sulfadiazine, 5 Gm. daily in divided dosage (2.5 Gm. twice daily), was employed universally. There were nine deaths in eighty pationts, a mortality rate of 11.25 per cent. The following year there was but one death in forty patients, and none in 1948. It is apparent that intravenous sulfadiazine and penicillin combined gave identically good results, as did the two combined with streptomycin, which was added in 1948.

TABLE XIII. PRESENT PERITONITIS REGIME

(1) Pantopon, ⅙ gr., q. 4 h. and p.r.n.
(2) Fowler's position
(3) Assist patient in getting out of bed to void
(4) Turn every hour
(5) Oxygen (nasal catheter) continuously (6 L. per minute)
(6) Continuous suction siphonage
(7) Streptomycin, ½ Gm., q. 3 h., and penicillin, 100,000 U., q. 3 h.
(To be given in same syringe by long intramuscular needle)
(8) 1,000 c.c. 5% glucose in normal saline solution intravenously daily at 8 A.M.
(9) 1,000 c.c. of protein hydrolysate intravenously daily at 4 P.M.
(10) 1,000 c.c. of 10% glucose in distilled water intravenously nightly at 12
(11) Surgical liquids, including carbonated drinks and coffee
(12) Warm mineral oil, 6 oz., q. 8 h. as rectal instillation
(13) Measure 24-hour fluid intake
(14) Measure 24-hour urinary output
(15) Vi-Syneral, 2 c.c. ampul, intramuscularly daily

500 c.c. of blood or 500 c.c. of dried plasma q. 3rd day as substitute for protein hydrolysate

Check: (1) R.B.C., W.B.C., Hgb., and hematocrit; (2) N.P.N., chlorides, and CO_2 on admission (recheck as indicated); (3) routine urinalysis.

The Peritonitis Regime.—In Table XIII are listed the established routine orders on our wards in all cases of peritonitis, excepting only the rare instance where a small abscess does not interfere with gastrointestinal normality. This regime brings into fruition all we have learned through trial and error, during the past three decades, of the values in physiologic and pathologic chemistry, chemotherapy, vitamin therapy, and the new antibiotics. It will be noted that intravenous sulfadiazine is no longer routinely used. Some staff surgeons have now substituted intravenous aureomycin, 100 mg. every six hours, in place of penicillin and streptomycin. By the time this paper is seen in print, perhaps one of the newer antibiotics may be substituted. After the first or second day, the third liter of intravenous fluid replacement is usually discontinued and often not used, if dehydration is not marked and the urine output is good (over 1,000 c.c. in the twenty-four-hour period).

SUMMARY

A comparison of mortality rates in the two past decades in patients with ruptured appendix with peritonitis is presented. The first decade, 1929 through 1938, comprised 444 cases with 134 deaths, an over-all mortality rate of 30.18 per cent. The mortality rate in the past decade, 1939 through 1948, in 541 cases and sixty-one deaths has been reduced to 11.27 per cent. The improvements represent a reduction of 18.9 per cent in the latter decade.

During the latter decade there were 3,396 cases of acute appendicitis of all grades with seventy-one deaths, an over-all mortality rate of 2.09 per cent. Among these were 2,680 cases of acute diffuse appendicitis with five deaths from all causes, a mortality rate of 0.18 per cent. There were 175 cases of acute gangrenous appendicitis with five deaths, a mortality rate of 2.85 per cent. There were 130 appendical abscesses included in the perforated series with a mortality rate of 6.15 per cent.

Approximately one-sixth (541 or 15.9 per cent) of all patients (3,396) admitted with a diagnosis of appendicitis in any grade had a ruptured appendix.

During the years 1947 and 1948 when penicillin and streptomycin were used intensively there was but one death in ninety-six patients (a patient not operated upon, in 1947), a mortality rate of 1.04 per cent.

CONCLUSIONS

With the very recent advent of chemotherapy, and the free use of blood and blood derivatives, and now the antibiotics, most noteworthy improvement in the mortality in peritonitis due to the ruptured appendix is an established fact. The better appreciation of the problems of fluid requirements, vitamin deficiencies, the introduction of suction-siphonage and intubation of the gastrointestinal tract, the improved methods of anesthesia, also have played their cumulative parts in enhancing the optimistic picture. Credit should be given, too, to the master surgeons who have taught us to operate in early perforation and to procrastinate, cautiously, in late rupture and in abscess formation.

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THE RELEASE OF VASOCONSTRICTION BY VASODILATOR DRUGS AND SYMPATHETIC NERVE BLOCK

An Experimental and Clinical Study

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THE release of vasoconstriction in an extremity is frequently a desirable therapeutic accomplishment in the treatment of peripheral vascular diseases. Vasospasm may be a purely functional disorder, as seen in the reflex sympathetic dystrophies and Raynaud's disease, or it may be a secondary phenomenon accompanying organic vascular disease such as arterial thrombosis, thromboangiitis obliterans, or thrombophlebitis. In the past few years much interest has been stimulated in the use of vasodilator drugs, and several new compounds have been released. On the basis of their pharmacologic action of vasodilatation a few have been employed quite extensively in the treatment of peripheral vascular diseases with the purpose of relieving vasospasm and increasing blood flow to one or more extremities.

There have been several reports in the literature supporting the clinical use of these drugs in the treatment of arteriosclerosis obliterans, Buerger's disease, Raynaud's disease, and others.[1-5] Most of these reports have been based primarily on subjective rather than objective evidence. Although the ability of these drugs to produce vasodilatation of normal blood vessels in some regions of the body is well accepted, little study has been made of their ability actually to release pathologic vasospasm in the extremities.

Previous studies in this laboratory of the drugs Priscoline,† papaverine, and nicotinic acid yielded information as to their relative potency as vasodilators in normal subjects in a room temperature of 78 to 80° F.[6] In the study reported here it was believed that the clinical state of vasospasm could best be simulated by producing vasoconstriction in normal individuals by exposure to a low environmental temperature. The purpose of the study was to elicit pertinent information as to the comparative efficacy of these drugs and peripheral sympathetic nerve block as agents for the release of this induced vasoconstriction. Such studies should give valuable information as to their possible beneficial use in the treatment of clinical vasospasm.

Sympathetic block has long been proved to be an efficacious method for releasing vasospasm, and for this reason the objective results obtained by this method were used as the standard against which the various drugs were evaluated. As an adjunct to these experiments, studies were also carried out on several patients with peripheral vascular disease.

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†Formerly known as Priscol (Ciba). The Priscoline used in this study was supplied by Ciba Pharmaceutical Products, Inc., Summit, N. J.

EXPERIMENTAL STUDIES

Normal young men and women with no evidence of cardiovascular disease were used as subjects. Blood flow changes in response to injections of various drugs and to sympathetic nerve block were measured by means of skin temperature variations, total toe volume, and mean pulse volume changes. The skin temperatures of the toes were determined by means of a Leeds-Northrup Speedomax automatically recording thermocouple. The Burch-Winsor plethysmograph was employed for measuring pulse volume and total toe volume changes. It is realized that these methods of study do not represent measurement of total blood flow to an entire extremity. In our earlier work with these drugs, muscle blood flow was measured indirectly by means of calculating the rate of removal of injected radioactive sodium from the gastrocnemius muscle. The rate of removal was shown to decrease uniformly after the injection of the various drugs. This decrease in muscle blood flow was so clearly demonstrated that it was believed to be unnecessary to include this technique in the present study. The techniques which were employed are an index of blood flow only to the skin and possibly the subcutaneous tissue.

The subjects were allowed to relax for thirty minutes in a constant-temperature room with the temperature at 78° F. Skin temperature measurements and plethysmographic results were obtained before cooling the room to 68° F. The temperature of the room was then maintained at this latter level with a variation from 66 to 70° F. for the remainder of the study. After cooling, measurements of blood flow were continued until constant results were obtained. One of the three drugs was then injected intravenously or a posterior tibial nerve block was performed, and the studies were continued for an additional thirty minutes. Total length of the individual study was approximately two to three hours.

RESULTS

The results of these experimental studies are presented graphically in Figs. 1, 2, and 3. For each subject in all four groups the maximal rise in skin temperatures of the toes was recorded (Fig. 1). The maximum increases in mean pulse volume and total toe volume were calculated from the Burch-Winsor plethysmographic tracings (Figs. 2 and 3).*

Nicotinic Acid.—Five subjects received 75 mg. of nicotinic acid intravenously. Moderate to marked subjective symptoms and objective evidence of facial flushing were noted in all the subjects. None of the three methods of study revealed any evidence of release of the induced vasoconstriction by this drug.

Papaverine.—Six subjects were studied. Three received 64 mg. and three, 32 mg. of papaverine intravenously. The full 64 mg. dose was not given to three individuals because of their marked subjective response, including dyspnea and apprehension, to the smaller dose. There was no difference in the objective response in the two groups. Again, in this group there was noted no significant

*In only three of the subjects receiving posterior tibial nerve block were continuous plethysmographic recordings made; thus total toe volume could be accurately calculated in only these three.

evidence of release from the induced vasoconstriction. Four of the individuals showed a brief, very slight rise in mean pulse volume and total toe volume, but none of the subjects showed a rise in skin temperature.

Priscoline.—Six subjects received 25 mg. of Priscoline intravenously. Moderate to marked subjective symptoms of tingling of the scalp, fullness of the head and nose, and body warmth gradually spreading into the extremities and followed by chilliness were noted by all the subjects. Contrary to the groups receiving the other two drugs, all the individuals receiving Priscoline exhibited definite evidence that their induced vasoconstriction had been released to some

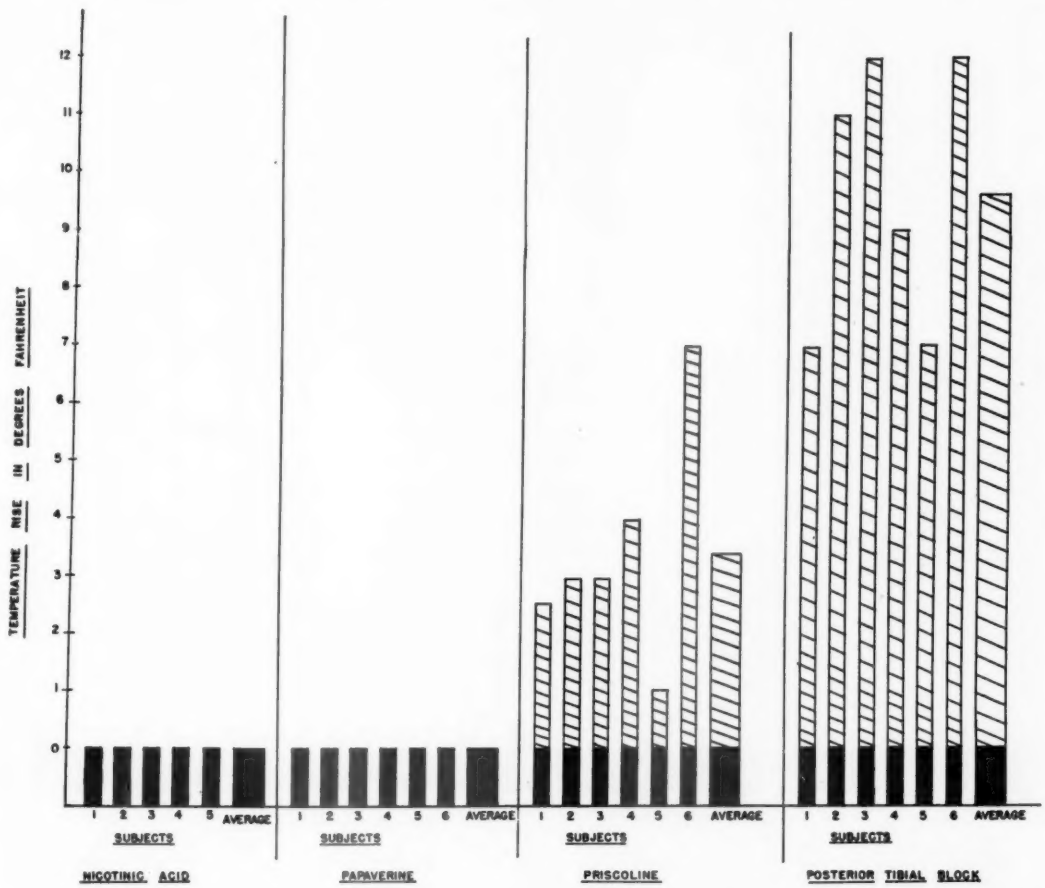

Fig. 1.—Comparative maximum increase in skin temperature produced by nicotinic acid, papaverine, Priscoline, and posterior tibial nerve block.

degree. They all showed rises in the skin temperatures of the toes, ranging from 1 to 7° (F.) and five of the six had a rise of 2.5° or higher. Similar increases were noted in the mean pulse volumes and the total toe volumes. The duration of these effects varied from ten minutes to over thirty minutes after injection of the drug.

Sympathetic Nerve Block.—Block of the posterior tibial nerve was carried out on six subjects with 10 to 20 c.c. of 1 per cent procaine. All demonstrated marked release of vasoconstriction. The skin temperature rise, total toe volume,

and mean pulse volume increase was uniformly much greater in this group than in the group that received Priscoline. Only one individual in the latter group exhibited a vasodilatation equal to that produced by posterior tibial nerve block.

Blood pressure and pulse determinations were taken before and at regular intervals following each procedure, but only insignificant changes were noted. No postural hypotension was demonstrated after any of the drugs. Most of the individuals used in the study received all three drugs on different occasions, which lends greater credence to the comparative results.

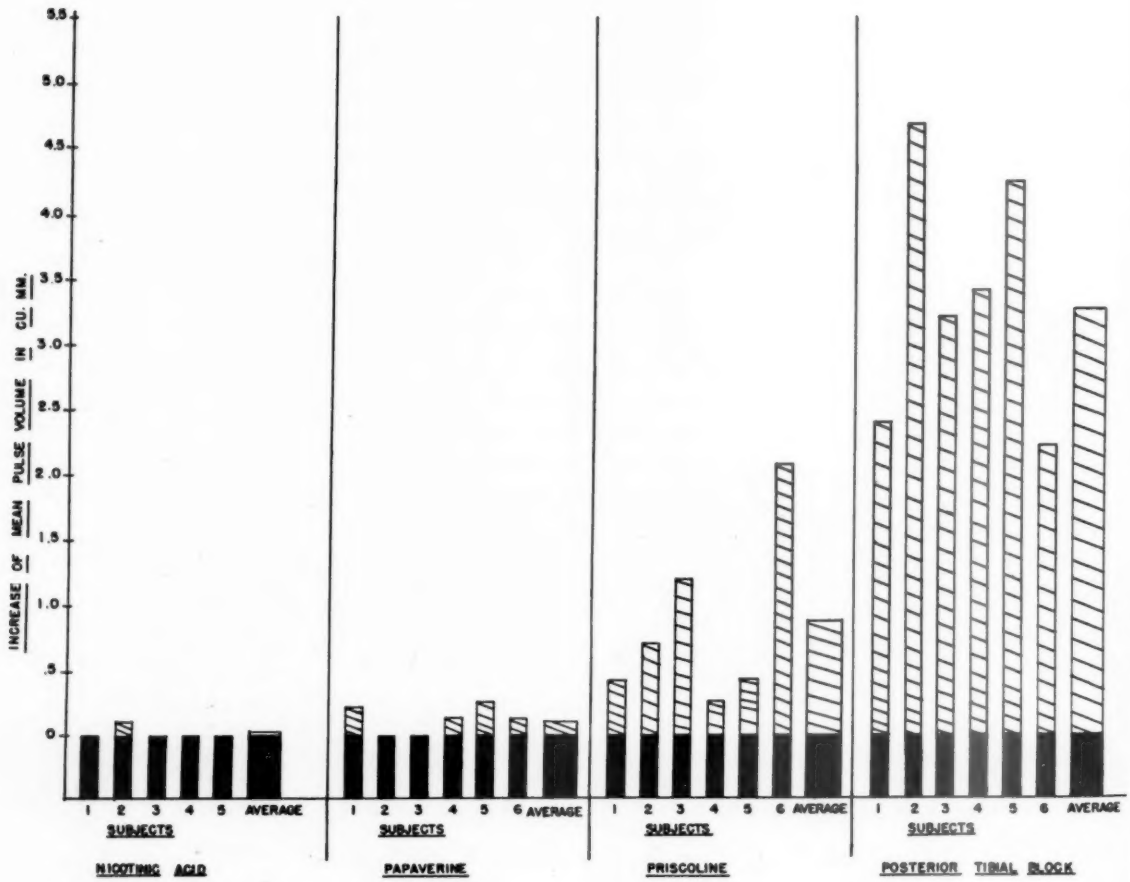

Fig. 2.—Comparative maximum increase in mean pulse volume produced by nicotinic acid, papaverine, Priscoline, and posterior tibial nerve block.

CLINICAL STUDIES

It was apparent from the results in the experimental study that nicotinic acid and papaverine were ineffective agents for the release of induced vasoconstriction. Priscoline was moderately effective, but was considerably less effective than sympathetic nerve block. These findings suggested the possible use of Priscoline in the treatment of clinical vasospasm. Accordingly, experimental studies were carried out on a small number of patients with peripheral vascular disease using the same techniques as were employed in the study of normal sub-

jects.* Priscoline was the only vasodilating agent used. Six patients were studied: four with organic occlusive arterial disease, and two with Raynaud's disease.

Of the four patients with organic disease, two had thromboangiitis obliterans: in one, pulsations were absent below the femoral region in one leg (the opposite leg was normal); in the other, there was involvement of the lower extremities bilaterally, and of one arm in which the radial pulse was absent (the opposite arm was normal). In the third patient, the popliteal artery had been ligated because of a traumatic arteriovenous fistula. The fourth patient had had an embolus to the left femoral artery with absent distal pulsations following a myocardial infarction. The two patients with Raynaud's disease presented classical signs and symptoms in all four extremities, most marked in the hands. Typical Raynaud's phenomena were demonstrated in both patients, and both had developed ulcerations and scleroderma of the fingers.

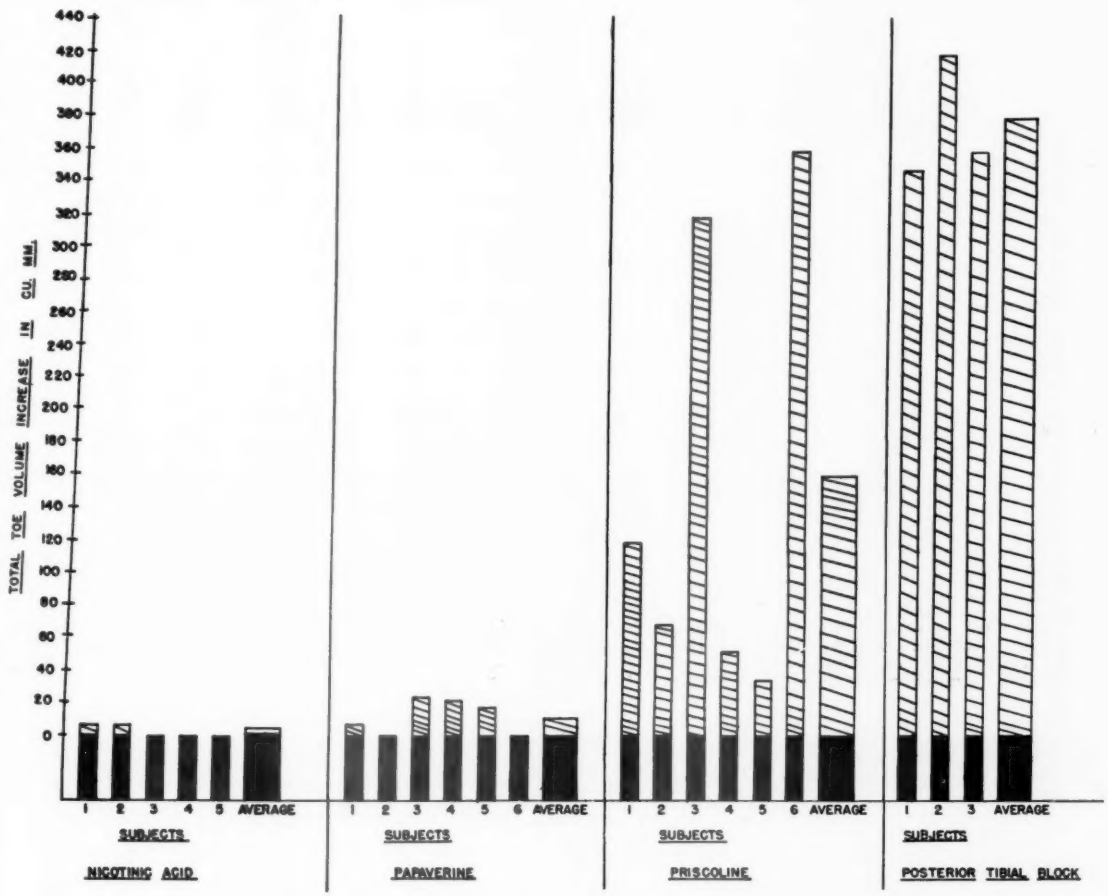

Fig. 3.—Comparative maximum increase in total toe volume produced by nicotinic acid, papaverine, Priscoline, and posterior tibial nerve block.

In the patients with organic occlusive arterial disease, skin surface temperatures and mean pulse volume changes of the digits were determined in the opposite normal extremity as well as in the diseased extremity. The two patients

*Total toe volume changes were not determined because it is impossible to record two leads constantly and simultaneously on the Burch-Winsor plethysmograph.

with Raynaud's disease did not have a normal contralateral extremity and no comparative study could be made. Priscoline, 25 to 50 mg., was injected intravenously after the control studies were completed.

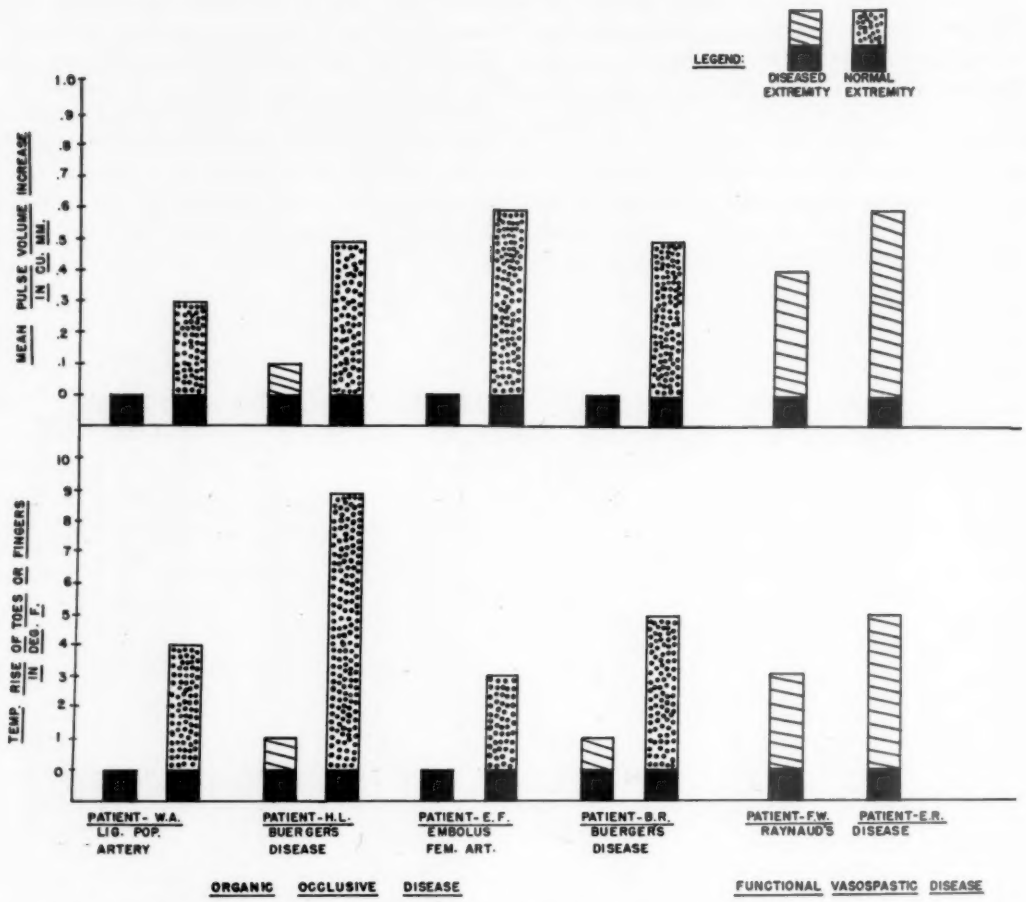

Fig. 4.—Comparative maximum increase in skin surface temperature and mean pulse volume of the digits of the normal and diseased extremities of four patients with organic occlusive arterial disease. The changes in the diseased digits of two patients with Raynaud's disease are also shown, but no normal extremity was available for comparison.

RESULTS

The results are presented graphically in Fig. 4. In the group with occlusive arterial disease, none of the patients demonstrated any significant vasodilatation in the diseased extremity as measured by rise in skin temperature or increase in mean pulse volume of the toes or fingers. The opposite normal extremity, however, exhibited considerable release of vasoconstriction. The two patients with Buerger's disease displayed a skin temperature rise of 1° in the diseased extremity, in comparison with an average rise of 7° in the normal extremity, and one of them showed a slight rise in mean pulse volume which was insignificant in comparison with the rise in the normal side. The other two patients exhibited no rise in either skin temperature or pulse volume in the diseased extremity, whereas the normal side displayed significant evidence of increased blood flow.

In Patient W. A., whose popliteal artery had been ligated, the skin surface temperature actually fell 1° during the thirty-minute period following the injection of Priscoline. All the patients noted a subjective sense of increased warmth in the normal extremity, but no change was noted in the extremity with arterial insufficiency.

The two patients with Raynaud's disease exhibited significant release of vasospasm in the fingers, and both noted a sensation of warmth which extended into the fingers and toes, and a complete relief of pain in the fingertips, which had been evoked by exposure to cold. There was an increase in the skin surface temperature and mean pulse volumes of the fingers.

DISCUSSION

The purpose of this study was to determine the ability of these commonly employed vasodilating drugs to release induced vasoconstriction as compared with sympathetic nerve block. It was believed that the intense vasoconstriction produced in the normal individual by exposure to cold would approximate the vasospastic conditions seen in clinical practice. This artificially produced state, however, is not exactly analogous to clinical conditions, for, in the latter, vasospasm is more commonly a localized phenomenon rather than a generalized one. It is apparent from these experimental results that nicotinic acid and papaverine are ineffective agents for the release of induced vasoconstriction. Priscoline is moderately effective but is considerably less effective than sympathetic nerve block. This study expands and confirms the work of Green and Ogle[7] who demonstrated, by the use of skin temperature measurements only, that Priscoline was able to release induced vasoconstriction in a majority of subjects.

This demonstration of the effectiveness of Priscoline in our experimental study led us to expand the study to include six patients with various forms of peripheral vascular disease. Four of the patients had organic arterial disease of an extremity. The opposite extremity was free of detectable vascular disease; it was considered normal and was used for comparison. The two other patients had Raynaud's disease without organic arterial change. In the first group it was clearly demonstrated that Priscoline was ineffective in increasing the blood flow to the distal portions of the involved extremity, whereas the normal side showed a response similar to that obtained in the group of normal subjects. These findings are similar to the findings of DeBakey and associates,[8] who demonstrated an actual decrease in blood flow in the diseased extremity following the injection of Etamon.

The failure of Priscoline in this group was not unexpected. In the presence of occlusion of the main vascular channels of an extremity, the circulation of blood is limited to small collateral vessels. These collateral channels may or may not be in a state of abnormal vasospasm. Because of their smaller size, their ability to produce a significant over-all increase in the capacity of the vascular bed by vasodilatation is considerably less than the ability of the normal vascular tree in the opposite extremity to increase its capacity.

Unlike sympathetic nerve block, the effects of which can be localized to a small area, the vasodilating action of Priscoline extends over the entire body

without selective action at any desired site. Its effectiveness as a vasodilator seems to be greatest in those vessels which are the most capable of increasing their caliber. Thus, Priscoline tends to vasodilate and increase blood flow throughout the body, including the normal extremity, but, due to the anatomic factors mentioned, with the consequent greater resistance to blood flow in the diseased extremities, little significant increase in flow is accomplished in that extremity where it is most desired.

If vasospasm is also present in the extremity which is the seat of organic occlusive disease, which is frequently the case, the drug would tend to be even less effective in producing the desired result of increasing blood flow to the vasospastic extremity. Priscoline, acting as a vasodilator, releases vasoconstrictor tone most effectively on those vessels which have the least tone at any given time, and its effects seem to be inversely proportional to the degree of vasomotor tone present. This is exemplified by the fact that in a previous study, normal subjects in an environmental temperature of 76 to 78° F., without induced vasoconstriction, exhibited uniformly a considerably greater rise in skin surface temperatures and mean pulse volumes than normal subjects in a cool environmental temperature displaying increased vasomotor tone, following the same dose of Priscoline.[6] In some instances, the blood flow to the diseased extremity may actually be reduced by the action of a generalized vasodilating drug such as Priscoline. This may have occurred in Patient W. A., who showed a fall in the skin temperature of the diseased extremity. This was pointed out and stressed by DeBakey and associates[8] in elaborating their theory of hemometakinesia.

It follows from this study that Priscoline, and probably all drugs which produce a generalized vasodilatation, are not to be recommended for the treatment of organic occlusive arterial disease, with or without the presence of superimposed vasospasm. Interruption of sympathetic nerves to an extremity, by procaine block or sympathectomy, is a far more effective agent for releasing vasoconstrictor tone and increasing blood flow to a diseased extremity. Its effects are predictable and well localized to the area involved.

The two patients with Raynaud's disease exhibited release of the vasoconstriction. In conditions primarily functional in nature, characterized by a generalized vasospasm without organic arterial disease, Priscoline may be a valuable therapeutic agent. It releases vasoconstriction and increases blood flow at least to the skin and subcutaneous tissues of the distal extremities. Continued administration of Priscoline should produce a fairly constant decrease in vasoconstrictor tone and thus alleviate the symptoms of coldness, numbness, and tingling, and prevent, or at least reduce, the frequency of the attacks of syncope in Raynaud's disease which are responsible for the eventual permanent trophic changes. From the results of these studies, it is our opinion that Priscoline deserves an extensive trial in the treatment of the generalized vasospastic disorders, including Raynaud's disease.

SUMMARY AND CONCLUSIONS

Experimental studies were conducted to evaluate the efficacy of three vasodilator drugs (nicotinic acid, papaverine, and Priscoline) and sympathetic nerve

block for the release of induced vasoconstriction. Nicotinic acid and papaverine were found to be completely ineffective. Priscoline was moderately effective, but it did not compare favorably with sympathetic nerve block. Nerve block produced the greatest vasodilatation and increase in blood flow.

Similar studies using Priscoline were carried out on six patients, four with organic occlusive arterial disease, and two with a functional vasospastic disorder. Priscoline proved to be ineffective as a therapeutic agent for releasing vasoconstriction and increasing blood flow in the former group, but was effective in the latter group.

It was concluded that Priscoline is not indicated for the treatment of organic occlusive arterial disease, but that it may be beneficial in the treatment of functional vasospastic disorders such as Raynaud's disease. It is believed that this drug is deserving of an extensive clinical trial in this latter group of patients.

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REPAIR OF OBTURATOR HERNIA WITH A TANTALUM GAUZE IMPLANT

Case Report

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THE obturator canal affords exit for the obturator vessels and nerve as they pass from the pelvis through the innominate bone. This region is one of those anatomic barrens that rarely comes to the attention of the general surgeon. The gynecologic surgeon occasionally undertakes a *tour de force* into this area during the course of an obturator lymph node dissection for pelvic malignancy. The orthopedic surgeon may make a rare foray into the region to perform an obturator neurectomy for the relief of intractable coxarthria. Aside from these procedures, the obturator region has seemed unimportant. However, there is an intrinsic lesion of the obturator canal, the obturator hernia, which the informed surgeon will wish to consider when confronted with the obscure symptom-complex of intestinal colic in the aged.

It is beyond the scope of this report to discuss the symptoms and findings of obturator hernia except to state that these features have been appreciated but seldom. Hence, the diagnosis of this condition rarely is made with accuracy, and the mortality is far out of proportion to the magnitude of the initial lesion. Obturator hernias are almost always strangulating by nature, and early diagnosis together with adequate surgical treatment are of the first magnitude in dealing with this entity.

The cause of the strangulation and the difficulties of surgical repair are appreciated when the anatomy of obturator hernia is considered. The obturator ring is formed above by the inferior margin of the pubic ramus, and below by the obturator membrane. It is through this foramen that the obturator artery, vein, and nerve leave the pelvis for their terminal distribution in the adductor region of the thigh and about the hip joint. The obturator canal is seldom more than 1 cm. in diameter and is consistantly occluded by a distinct collection of preperitoneal fat.[1] It is the occasional deficiency of this lipomatous barricade which allows a peritoneal process to dip into the obturator canal. This canal is actually a cul-de-sac which ends beneath the pectineus muscle. Three varieties of obturator hernia have been described,[2, 7] depending upon the relationship of the sac to the obturator externus muscle, that is, whether it passes over, through, or beneath the muscle. Regardless of the type of hernia present, the sac lies deeply in the adductor region and rarely gives external evidence of its presence in the thigh.

As stated, eventual strangulation is almost the rule in obturator hernia. The unyielding nature of the internal opening of the obturator canal, bounded above by a bony wall and below by the sharp edge of a firm, inelastic membrane, makes apparent the site and cause of the strangulation.

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The rigidity of these osseoligamentous structures also accounts for the difficulties experienced in the adequate repair of obturator hernia, and explains the great frequency of hernial recurrence.[3, 8, 11, 13] The fundamental concepts of adequate herniorrhaphy require the fulfillment of two criteria: (1) the removal of the hernial sac, and (2) the obliteration of the hernial defect. Obturator herniorrhaphy is no exception to these rules. The removal of the sac usually has been simple; the closure of the actual hernial defect has not been tried or has proved impossible in most cases. Obviously, the obturator foramen is not amenable to closure by suturing. The usual "closure" of this defect has been obtained by suturing the hernial sac as a pad over the ring. The effectiveness of this method is open to some doubt. The pelvic peritoneum in this region is very lax, and the rigid obturator ring can only invite a recurrence when the flimsy peritoneum is stretched over it as a barricade. Some surgeons[4, 9, 12] have used flaps cut from the pectineus muscle and sutured as a plug to the periosteum of the pubic ramus. Such a procedure seems difficult, requiring a combined type of operation with exposure by both intraperitoneal and adductor routes. Short[8] reoperated on a recurrent obturator hernia and ingeniously fashioned a plug of costal cartilage which was inserted into the obturator ring. It is thus apparent that various surgeons occasionally have attempted to improve upon the "routine" type of obturator herniorrhaphy by various autoplastic barricades. However, the advent of tantalum gauze into the field of herniorrhaphy has placed a more ready and simple means of closure in the surgeon's hands.[10]

It has been shown that tantalum gauze, a finely woven mesh of tantalum wire, may be safely implanted in the tissues of the abdominal wall.[5, 6, 10] Here it acts as a biologically inert foreign body and is rapidly ensheathed in dense collagenous tissue. Such an implant, together with its fibrous tissue envelope, forms a sturdy buttress over any hernial defect when local tissue deficiency does not allow a satisfactory closure by more conventional means. The application of such a tantalum gauze implant to obturator herniorrhaphy seemed logical and was attempted in one case.

CASE REPORT

The patient, F. G., was an emaciated man, aged 69 years. He had been troubled, over a four-year period, with bouts of severe, intermittent intestinal colic. These attacks coincided with the onset of severe right coxarthria and shooting pains along the distribution of the right obturator nerve. These bouts he could relieve by lying down, flexing the right thigh, and massaging the right groin. A previous diagnosis of femoral hernia had been made, though none was apparent. A suprapubic prostatectomy also failed to relieve his distress, and the problem was subsequently considered as "functional."

He was referred to me on July 31, 1945. His general physical build, his story of true intermittent intestinal colic, and the concomitant onset of pain along the distribution of the obturator nerve (Howship-Romberg sign) led to a diagnosis of intermittent, right obturator enterocele, and operation was advised. Laparotomy was done on Sept. 1, 1945, using a low, right pararectus incision (Fig. 1, *a*). This type of incision was chosen as one which would give adequate access to the right obturator region, and also simplify the repair of a small diffuse ventral hernia present at the right extremity of the old transverse suprapubic scar. As the patient was placed in Trendelenburg position, the right obturator hernia was at once

apparent. There was no content in the hernial sac and the sharp, rigid obturator ring just admitted the tip of the little finger (Fig. 1, *b*). Inspection of the terminal ileum revealed multiple areas of serosal scarring and puckering, obviously the sites of repeated trauma. An Allis forceps was slipped into the obturator canal; the fundus of the sac was engaged in its jaws and inverted into the abdomen with moderate traction (Fig. 1, *c*). There was a lipomatous accumulation of fat at its tip. The sac and lipoma constituted a mass 5 cm. in length. The sac was excised and, with the peritoneum open, the obturator vessels and

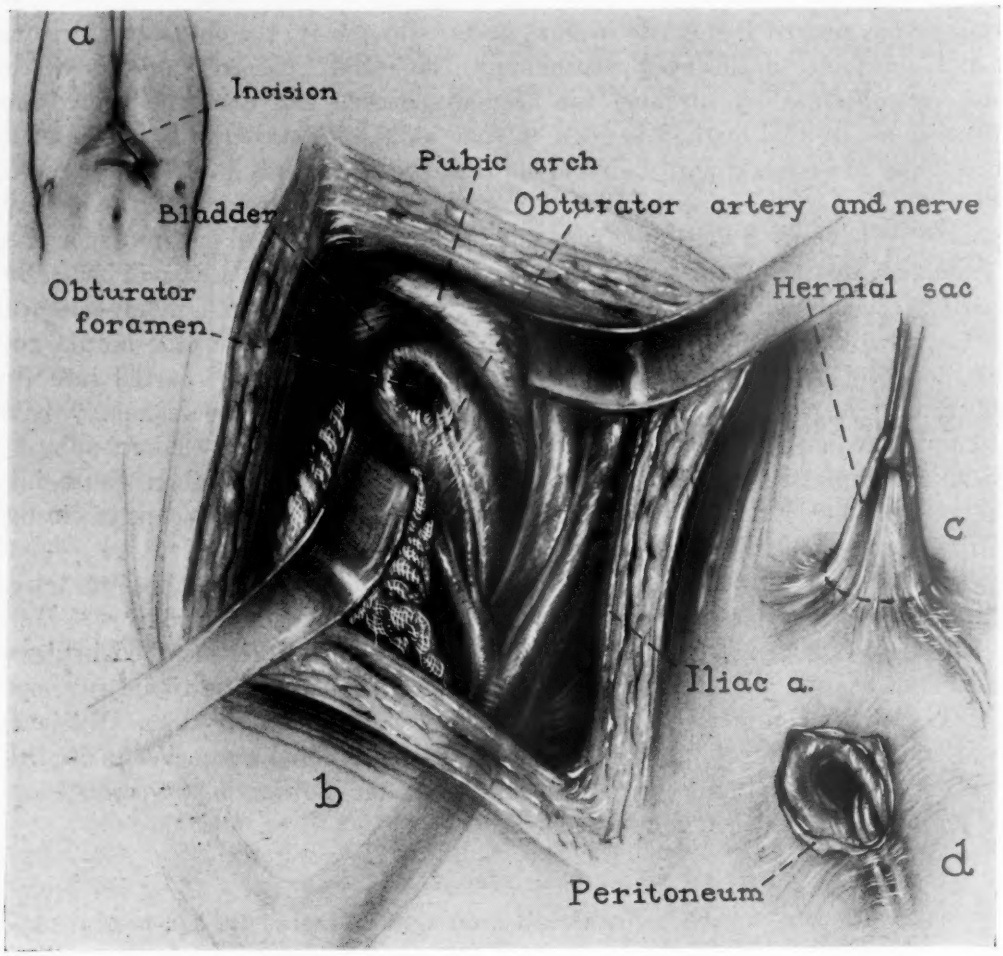

Fig. 1.

nerve could be identified coursing over the inferolateral margin of the obturator ring (Fig. 1, *d*). It was obviously impossible to close a defect with such rigid margins by simple suture, and it was decided to barricade the obturator ring by means of a tantalum gauze implant. A piece of tantalum gauze, composed of 2 mil* wire in a 100 by 100 weave,† was cut to 3.5 by 3.5 cm. dimensions. The free margins of this implant were folded under for approximately 0.5 cm., leaving a smooth, atraumatic edge and producing a quadruple thickness of gauze at each corner (Fig. 2,*a*). This implant was sutured in place by means of a single stitch of monofilament 5 mil tantalum wire in each corner. The two superior sutures were placed in the periosteum of the pubic ramus; the two inferior sutures were taken in the obturator membrane (Fig. 2, *b*). The obturator vessels and nerve ran beneath the

*A mil is 0.001 inch, used as a measure of wire diameter.

†This weave of 2 mil wire was experimental and is not available. The usual 50 by 50 weave of 3 mil wire would have served the purpose equally well.

lateral margin of the implant without compression. The peritoneum was closed over the tantalum gauze with sutures of 0 chromic catgut (Fig. 2, *c*). The entire repair did not require five minutes. The abdominal incision was closed with interrupted sutures of alloy stainless steel wire, imbricating the layers at the site of the ventral hernia. The postoperative course was uneventful and the patient was dismissed from the hospital on the tenth postoperative day. The patient has been seen occasionally since the operation, the most recent visit being on March 15, 1948, at which time he remained free of the former distress. His local physician assured me more than one year later that the result was still completely satisfactory. Stereoscopic x-ray views of the obturator region reveal the tantalum gauze implant fitting like a shallow dish over the region of the obturator canal. The gauze has remained intact, probably because lack of motion or stress in this region has not led to work-hardening and fracture of the implant. The abdominal incision is well healed, without recurrence of the ventral hernia.

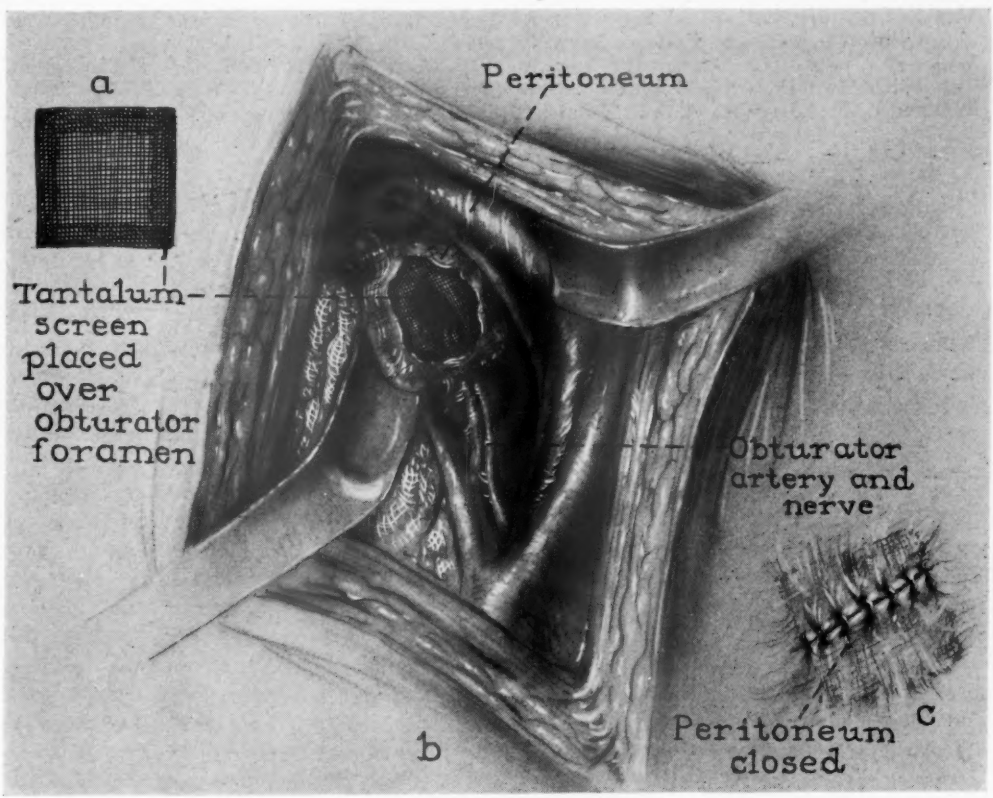

Fig. 2.

DISCUSSION AND CONCLUSIONS

Tantalum gauze has proved an effective means of closing hernial defects where tissue deficiency or the type of defect has made a satisfactory autoplastic closure impossible. The use of such a tantalum gauze implant in this case of obturator hernia has simply and effectively barricaded the rigid internal obturator opening for four years, and there is every reason to assume that this satisfactory result will be permanent.

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Case Reports

LACERATIONS OF THE JEJUNUM BY A TAUT STRING

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THE lacerations of the jejunum to be reported occurred in a 9-year-old white girl. The lacerations were produced by a displaced esophageal dilating string which was drawn into the jejunum by the broken flange of a gastrostomy tube. The string traversed the esophagus and nasopharynx, and was anchored at the external abdominal wall (Fig. 1). A similar case was not encountered in the literature.

CASE REPORT

A 9-year-old white girl was admitted to the Crippled Childrens Hospital Dec. 26, 1948, with abdominal pain. In March, 1944, a gastrostomy was performed to permit esophageal dilatations for a stricture from lye burns received in 1943.

Five days prior to admission the gastrostomy tube was dislodged from the operative stoma and the flange was missing. The onset of upper abdominal pain three days before admission was followed by the regurgitation of gastrointestinal content from the gastrostomy stoma. Shortly thereafter, obstipation and dehydration developed. A similar episode was experienced in December, 1945, but no relevant symptoms developed, and within a few days the flange was recovered from a stool specimen.

Examination revealed an acutely ill, dehydrated patient. A continuous discharge of thin, green-staining material was observed at the gastrostomy site. The temperature was 100° F., pulse rate 105. The respiratory rate was 16 and the blood pressure 105-60. A string, entering the left nasal orifice, traversed the nasopharynx and esophagus and extruded from the gastrostomy stoma. It could not be withdrawn by gentle manipulaion. The heart and lungs were normal. The abdomen was slightly distended, moderately tender, and peristalsis was present. An indefinite tender mass was palpable in the right lower quadrant. Digital examination of the stomach through the gastrostomy stoma did not reveal the missing flange. Rectal examination was negative. The hemoglobin was 12.5 Gm. per cent; red blood cell count was 4,890,000; the white blood cell count was 19,800 with 87 per cent neutrophils. Specific gravity of the urine was 1.025. Sugar, albumin, and cells were not demonstrable. The blood chloride determination was 360 mg. per cent.

After intravenous infusion of saline solution and plasma, the child's condition improved. In addition to regurgitation from the gastrostomy stoma, vomiting persisted. Dark blood was soon recognized in the material from both sites. Several x-ray examinations of the abdomen revealed no definite evidence of progression of the flange. The signs and symptoms suggested a high intestinal obstruction.

At operation successive loops of jejunum, distal to the ligament of Treitz, were held in intimate contact by a plastic exudate. After separation of these loops, the gastrostomy flange was located at a point three feet from the ligament of Treitz. Proximal to the flange, numerous lacerations of the jejunum were present. The loop of the dilating string was rendered taut by the progressing flange. The two segments of string extruding from the individual lacerations in the jejunum were embedded in the corresponding portions of mesentery. The most proximal laceration was at the ligament of Treitz. The lacerations were

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jagged and there was considerable injury to the adjacent mesentery. The duodenum appeared intact and manual exploration disclosed no abnormalities. Resection was deemed advisable and an end-to-end anastomosis was performed.

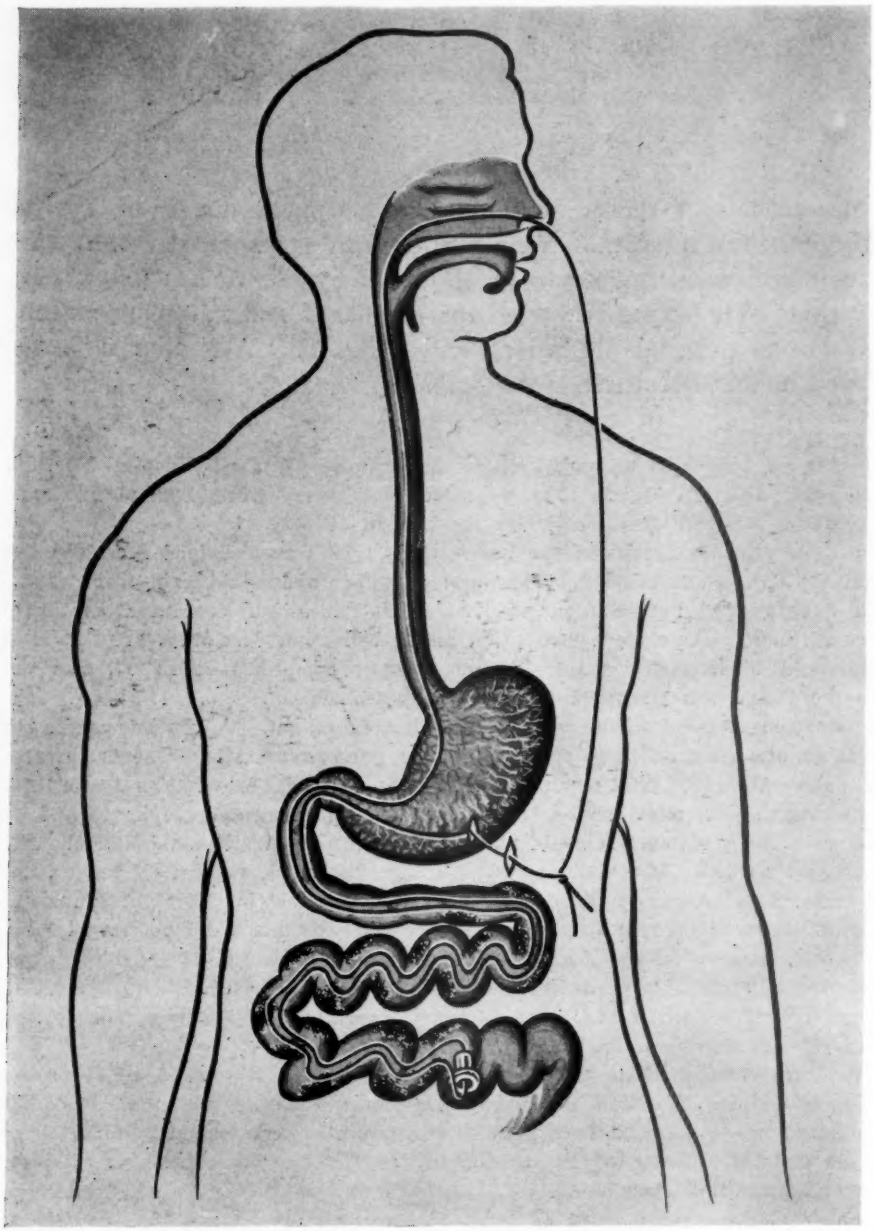

Fig. 1.—Position of broken flange and esophageal dilating string.

The specimen consisted of a segment of jejunum 64 cm. long and 4 cm. in diameter, and 4 cm. of attached mesentery (Fig. 2). In the mesentery there were various-sized lymph nodes up to 1 cm. in diameter. Along the length of bowel there were twelve jagged rents, ranging from 1 to 8 cm. in length, with everted margins. Some of these rents were along the mesenteric border. The edges were hemorrhagic and the mucosa was dark red.

Complete perforation was obvious at each laceration site. Serosal surfaces elsewhere were dull and dark red.

Postoperatively, the patient was critically ill and demonstrated no definite improvement for four weeks. Three episodes of massive gastrointestinal hemorrhage occurred during this period. Repeated blood transfusions and parenteral fluids were given. Large doses of penicillin were administered for four weeks. Other measures included coagulant drugs by mouth and parenterally. After cessation of the hemorrhage, recovery was uneventful and she was discharged on the fiftieth postoperative day.

Five months following surgery, esophageal dilatations were no longer necessary. She was asymptomatic and the nutritional status was normal. The gastrostomy wound was closed and the postoperative course was uneventful.

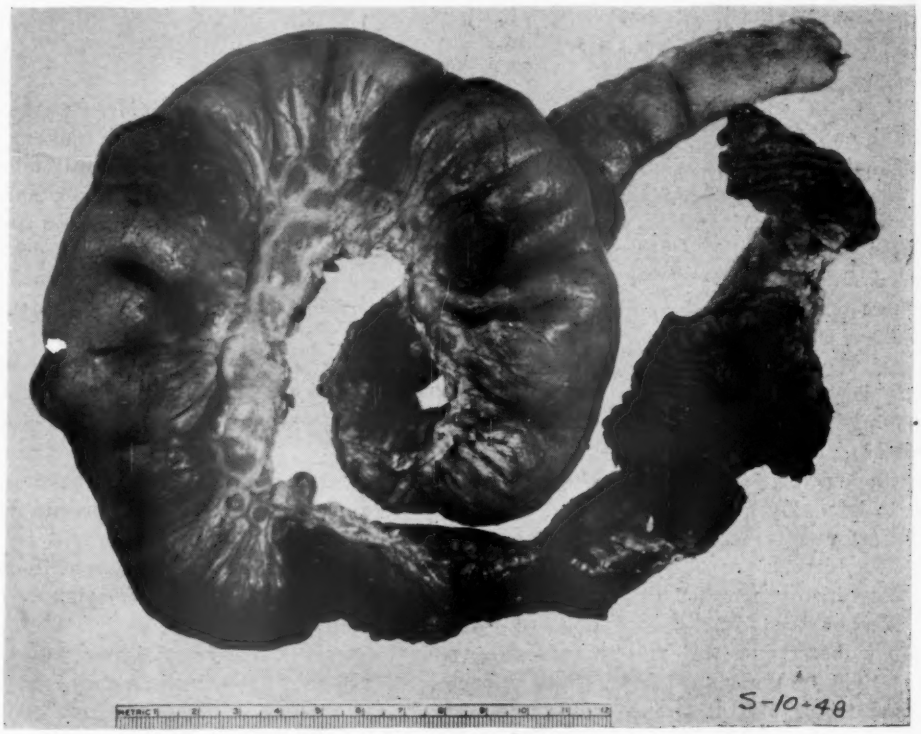

Fig. 2.—A 64 cm. segment of jejunum with twelve jagged lacerations.

COMMENT

A case of multiple lacerations of the jejunum in a 9-year-old white girl is reported. A displaced esophageal dilating string was the offending agent. A loop of the string had been drawn into the jejunum by a broken gastrostomy tube flange. Peristaltic propulsion of this flange created a sawlike action of the string against the jejunal wall and produced the lacerations. This case emphasizes the necessity for the immediate examination of any patient in whom a gastrostomy tube has been broken or dislodged. Parents should be made aware of this hazard.

DUODENAL OBSTRUCTION, SITUS INVERSUS, AND NONROTATION OF THE COLON

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CONGENITAL abnormalities are often multiple in occurrence. Appearing singly, they may not attract special attention, but the combination of several rare congenital anomalies is usually interesting.

It seems worth while to report the case history of a child who had duodenal obstruction, incomplete situs inversus, nonrotation of the colon, and probably an absence of the spleen.

CASE REPORT

E. C., a colored female, 14 months of age, was admitted to Children's Memorial Hospital on May 12, 1948. The admission history was obtained from her mother, who stated that vomiting had been present since birth but had never been severe until the child was 6 months of age. At that time an attack occurred which lasted one week and was characterized by projectile vomiting. A physician saw the infant and stated that an obstruction was present which might require surgical intervention. However, the attack gradually subsided and little difficulty was subsequently experienced, although at no time since birth had more than three or four days elapsed without the occurrence of vomiting. Constipation was also a constant symptom.

On May 8, 1948, she began to vomit three or four times daily immediately after eating. During her feedings food flowed from the mouth. After one or two days she refused everything except milk and strained food. The vomitus was either yellowish green or definitely green in color.

On May 11, 1948, the mother observed large waves in the upper abdomen passing from right to left. Marked restlessness and irritability were also noted. No bowel evacuation had occurred for several days before the patient's entrance to the hospital.

The birth history, developmental history, and family history were noncontributory.

Past history revealed that the child had measles in March, 1948, with complete recovery.

The physical examination disclosed certain pertinent findings.

The heart was outlined by percussion and the area of cardiac dullness was located to the right of the sternum. The lateral border of cardiac dullness was in the right nipple line and the heart tones were heard best over the right anterior thoracic region.

The abdomen was somewhat protuberant. Large peristaltic waves were visible in the upper abdomen passing from right to left. Percussion outlined the liver on the left side, although a small portion could be palpated in the midline. An umbilical hernia was present through a defect about 1.5 cm. in diameter.

The laboratory examination of the blood disclosed an erythrocyte count of 4,700,000, leucocyte count of 13,300, and hemoglobin of 14 Gm. The urine showed four to six white blood cells with some clumping in the high-power field.

Roentgenographic evidence of duodenal obstruction was obtained. The duodenum was seen to be in the left upper quadrant of the abdomen and the stomach in the right upper quadrant with a reversal of the normal relationship (Fig. 1). At the same time a chest roentgenogram showed the heart to be situated largely in the right thoracic cavity (Fig. 2).

The electrocardiogram also indicated dextrocardia, which confirmed the clinical and roentgenographic evidence.

From May 12 to 15, 1948, daily stomach aspirations were performed and dehydration was corrected with intravenous fluids. Her condition improved and on May 15, 1948, she was operated upon.

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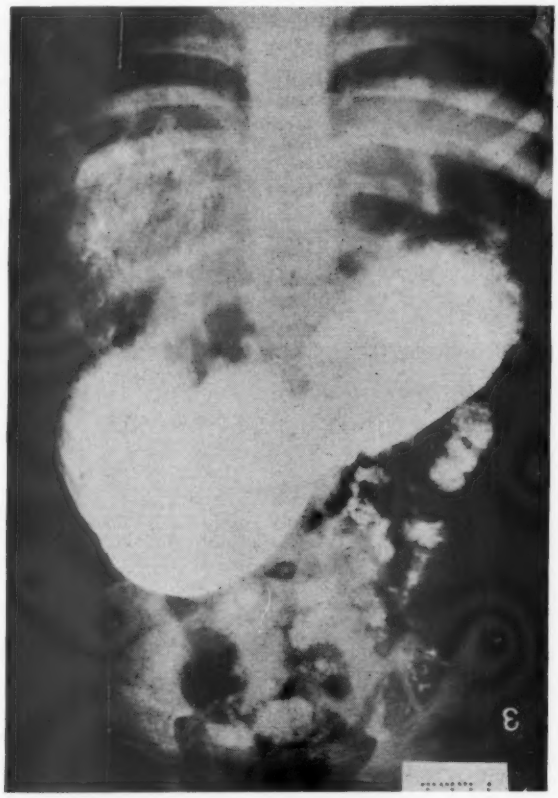

Fig. 1.—Roentgenogram showing greatly dilated duodenum in left upper abdominal cavity.

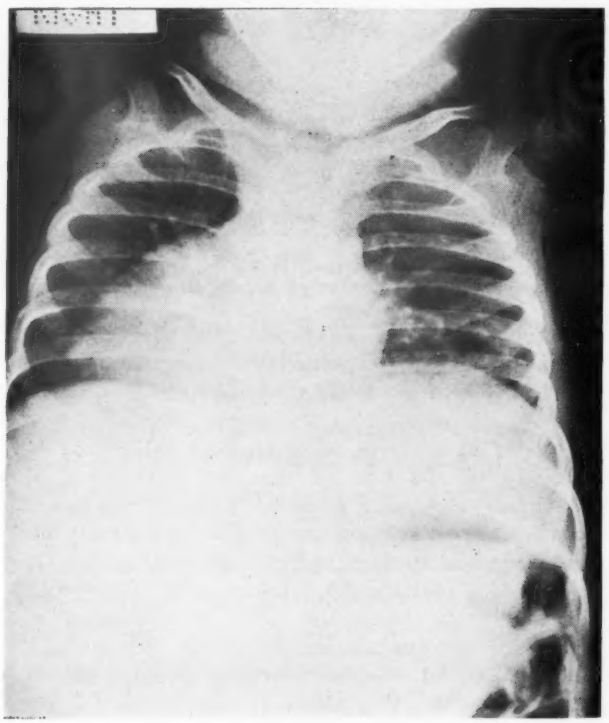

Fig. 2.—Chest roentgenogram showing dextrocardia.

At operation the abdomen was opened through a vertical left paramedian incision so that the duodenum would be readily accessible. The esophageal hiatus was located to the right of the vertebral column. The stomach was found mainly in the upper right abdomen with the curvatures passing from above downward, from right to left. The duodenum and the head of the pancreas were lying to the left of the vertebral column. The large lobe of the liver occupied the left upper quadrant, with the gall bladder, bile ducts, falciform ligament, and round ligament situated to the left of the midline. The smaller hepatic lobe (normally the left) was in the right upper abdominal quadrant. The ascending colon was lying along the right parietal wall, and as it reached the hepatic flexure the transverse colon dipped sharply downward and then upward again, so that the splenic flexure and descending colon also were in the right half of the abdominal cavity (Fig. 3).

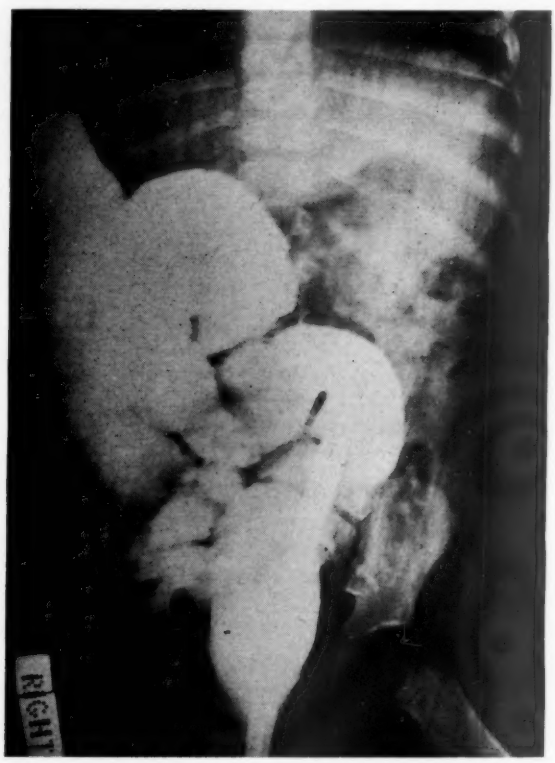

Fig. 3.—Barium in colon showing most of large intestine in the right half of the abdominal cavity (postoperative roentgenogram).

The kidneys were in their usual locations. An effort was made to identify the spleen, but after careful search by inspection and palpation of both upper abdominal quadrants no spleen could could be found. A search for accessory spleens was also unrewarded.

The pathology consisted of adhesive bands on both medial and lateral surfaces of the duodenum at approximately the junction of the first and second portions. A duodenal kink was evident in this area. Extending from the posterior and lateral parietal peritoneum of the left upper abdomen, a definite peritoneal fold was seen which terminated on the lateral duodenal border. Proximal to the kinked, constricted, duodenal area the intestine and stomach were greatly dilated. Distally the intestine was collapsed.

All the adhesions and folds around the constricted duodenal area were divided. A rubber shod clamp was placed on the mid-portion of the stomach and distal to the clamp gentle manual pressure was exerted. Gastric contents passed easily through the previously constricted area. The abdomen was then closed.

Following the operation the child seemed to be progressing in a satisfactory manner until May 20, 1948. At that time she began to vomit all her feedings and aspiration of the stomach showed evidence of gastric retention. Plasma, whole blood, and intravenous fluids were given for the next few days in the hope that the evidence of high obstruction would disappear. The obstruction persisted and it was felt that surgical intervention was again necessary.

On May 24, 1948, a second laparotomy was performed through the old incision. Numerous adhesions were present at the site of the previously obstructed area, with additional ones which had formed around the distal portion of the stomach causing adherence to the liver. Some of the adhesions were freed and an anterior gastrojejunostomy was performed. An attempt to find the spleen or accessory spleens again was unsuccessful.

After the second operation recovery was rapid and uncomplicated except for an episode of acute pharyngitis. The patient left the hospital on June 9, 1948, and has been seen a number of times in the outpatient clinic since that date. Growth and development are normal and no symptoms referable to the gastrointestinal tract have been observed.

DISCUSSION

Each of the four anomalies in this patient will be discussed separately; namely, (1) situs inversus, (2) nonrotation of the colon, (3) duodenal stenosis, and (4) absence of the spleen.

Situs Inversus.—This malformation is manifested by transposition of the various organs as in a mirror image. It may affect all or part of the internal organs. It is an extreme type of symmetry reversal. Left handedness and counterclockwise hair whorl on the crown are other more familiar but milder expressions of the same tendency.[1] Dextrocardia may occur alone as may reversal of the relationship of the abdominal viscera.

The cause is theoretical. Stockard[10] and Newman[9] produced reversal in experimental embryos (starfish) by changing the temperature or the oxygen supply. Cockayne[4] believed the cause to be hereditary in origin, a recessive gene being responsible.

Situs inversus is often associated with a variety of other congenital anomalies. Congenital cardiac malformations, absence of the spleen, duodenal atresia, imperforate anus, Meckel's diverticulum, and absence of the appendix have all been included in reports as accompanying developmental defects. Our case is an example of situs inversus with several associated defects.

The occurrence of situs inversus has been recognized for many years. According to Lineback,[7] Fabricus described a case of reversed liver and spleen in a human body in 1600 A.D. Vehsemeyer[11] in 1897 demonstrated situs inversus by roentgenography. Blegen[2] collected data on the incidence of situs inversus from a large number of publications. Based on roentgen-ray examinations and hospital admissions of 463,950 patients, the incidence was 1 in 6,587. In 117,000 autopsies there were twenty instances of situs inversus, or 1 in 6,164. Mayo and Rice[8] stated that 76 of 1,551,047 patients at the Mayo Clinic from 1910 through 1947 had complete situs inversus on the basis of physical or roentgenologic observation or both, an incidence of about 1 in 20,000.

At the Children's Memorial Hospital from 1937 to 1949, there were 170,153 patients examined. Of these patients, 38 exhibited situs inversus, an incidence of 1 in 4,477. Of this number 17 involved the thoracoabdominal

organs and 21 showed dextrocardia only. The more frequent occurrence of situs inversus in a children's hospital is to be expected as this anomaly is likely to be discovered in childhood.

Nonrotation.—When nonrotation of the intestine occurs, the jejunoileum lies on the right side and the colon on the left side.[1] In our case, because of the situs inversus, the jejunoileum was on the left side of the abdominal cavity and the entire colon on the right side. Had normal rotation occurred in association with the situs inversus, the appendix and ileocolic juncture would have been in the left lower abdominal quadrant and the colon distributed in a mirror image of the usual pattern.

Normally, intestinal rotation occurs in three stages.[5] At about the fifth to the seventh week of fetal life the midgut pushes through the umbilical orifice and remains out for five weeks, elongating and undergoing a counterclockwise rotation of about 180 degrees around the superior mesenteric artery.[1] This is the first stage. It then returns to the abdomen (second stage) and continues rotating, finally becoming stabilized (third stage). If it fails to rotate during its period of extrusion, nonrotation exists. Varying degrees of malrotation have also been described, usually resulting from either incomplete rotation or a clockwise rather than an anticlockwise rotation of the intestine around the superior mesenteric artery.

Duodenal Stenosis.—Stenosis of the duodenum, as in other portions of the small intestine, may be due to a failure of the intestinal lumen to recanalize properly after the second fetal month when the intestinal tube passes through its solid stage. The solid stage is due to epithelial proliferation and the intestinal lumen is filled with epithelial concrescences. During the process of recanalization an epithelial septum may persist completely or partially. If complete, an atresia results; if partial, a stenosis.

Stenosis may also be due to peritoneal bands or adhesions. This was apparently the cause in our patient. These bands are due to either fetal peritonitis or abnormal development and are often associated with malrotations.

Infection extending along the umbilical vein to the hilum of the liver and thence to the duodenum may cause the formation of obstructing adhesions in some instances.[3]

Abnormal development may account for peritoneal folds which are smooth and apparently not the result of an infectious process. Our patient possessed, in addition to adhesions, a definite fold or band of peritoneum extending from the junction of the first and second portions of the duodenum at the site of obstruction to the lateral body wall in the left upper abdominal quadrant. This fold was attached to the antimesenteric surface of the duodenum and was the main cause of the stenosis.

As far as we know, the reason for the occurrence of such a fold of peritoneum has never been determined. To account for this anatomic variation a theoretical explanation occurred to us.

If a portion of the ventral mesentery persisted it could produce a fold such as that found in our patient. As the duodenum develops and undergoes

rotation this persistent portion of the ventral mesentery could conceivably fix, compress, or kink a segment of duodenum and ultimately produce a stenosis.

Normally, in the upper abdomen, the falciform ligament of the liver and the anterior wall of the omental bursa represent the persistent portion of the ventral mesentery. The anterior wall of the omental bursa is divided into the hepatogastric and hepatoduodenal ligaments.[1] The hepatoduodenal ligament ends caudally at the epiploic foramen. If it extended a short distance distally from this point a fold such as we encountered could result.

Ladd and Gross[6] found stenosis in the duodenum in eleven of their twenty-two cases of intestinal or colonic stenosis. No case of multiple stenosis was found. This is in contrast to atresias where more than one is often found. They stated that the higher the obstruction, the better the outlook. Seven of their eleven patients with duodenal stenosis recovered.

Absence of the Spleen.—Congenital absence of the spleen is an unusual finding that is wholly compatible with life. It may be discovered accidentally or at autopsy. Cockayne[4] mentioned three cases of absence of the spleen accompanying transposition of the viscera.

In our patient the spleen could not be demonstrated at either operation. Accessory spleens were not found. It is probably safe to assume that the spleen was absent. Whether or not a relationship between situs inversus and absence of the spleen exists is an interesting speculation.

SUMMARY

A case history is reported of a patient who had duodenal obstruction (stenosis), situs inversus, nonrotation of the colon, and probably an absence of the spleen. Two operations were necessary for relief of the duodenal obstruction. Recovery was satisfactory and apparently complete.

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LONG-STANDING ASYMPTOMATIC PARTIAL INTESTINAL OBSTRUCTION CAUSED BY FOREIGN BODY AND STRICTURE OF THE ILEUM

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THE types of foreign bodies which have been reported as present in the gastrointestinal tract of man are almost infinite in their variety. Some of the ingested foreign bodies reported in the literature are knives, forks, spoons, pencils, razor blades, nails, screws, coins, buttons, toothpicks, bones of all varieties, needles, radium capsules, and beer mug handles.[1-15] Some of these have resulted in perforation of the gastrointestinal tract and of diverticula.[16, 17] Wolfler and Lieblein's monograph "Die Fremdkorper des Magen-Darmkanals des Menschen"[18] covered the field so thoroughly that we feel it unnecessary to review the earlier literature. Recently MacManus[17] classified swallowed foreign bodies into three main types: metallic objects, bones, and wood splinters. The first two groups are of equal incidence and constitute the great majority.

In most instances the foreign bodies are soon passed by natural routes or are removed by the surgeon. Smith[19] stated that it is most unusual for foreign bodies to remain in the intestinal tract for long periods of time, and reported a case in which twenty years elapsed between the ingestion and ejection of a darning needle. Other protracted stays are reported by the following authors:

LeGendre:[20] A fork was passed by rectum 456 days after being swallowed.

Dufresne:[21] A saber blade 10½ inches by 1 inch was recovered from an abscess one year after swallowing.

Cerne:[22] A spoon was recovered by gastrotomy after ten years.

Wakefield:[23] Seven coins were passed by rectum after twenty months.

Nuttweg:[24] Cherry stones were recovered by stomach tube, and also were passed by rectum, two and one-half years after ingestion.

The general symptoms are: a sense of weight, interference with appetite, nausea, vomiting, pain, and weight loss.[1] The diagnosis is usually made from the history and from x-ray examinations. Occasionally the foreign body can be palpated.

We wish to present a case in which a foreign body (fruit pit) was present in the ileum for at least four and one-half years, and which was hindered in its passage to the outer world by a stricture at the site of a Meckel's diverticulum.

CASE REPORT

The patient, a 60-year-old railroad detective, was first admitted to the Batavia Veterans Hospital in October, 1943. His complaint at that time was of constipation and epigastric pain, which had been present intermittently since 1918. The pain could be relieved by milk and antacids. Gastrointestinal x-ray pictures taken Oct. 7, 1943, showed two areas of nar-

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rowing in the terminal ileum, which were interpreted as regional ileitis. A second series taken one week later showed similar findings and, in addition, an oval density 2.5 cm. in its greatest diameter was seen in the right lower abdomen. This was thought to be a calcified lymph node.

He was readmitted in November, 1945, with the same complaints. X-ray studies at this time showed no evidence of ileitis. The oval density was still present. A diagnosis of chronic gastric hyperacidity was made and the patient placed on an ulcer regime. He was discharged after one month's hospitalization.

The patient presented a similar story on his third hospitalization in December, 1947, with a few additional complaints. He had lost twenty pounds in the past six months, and had become very nervous and easily upset. X-ray studies at this time demonstrated the oval density to be in the ileum and an area of mild stenosis was seen immediately distal to it. The roentgenologist considered the shadow to be a prune pit. On further questioning the patient admitted to a fondness for prunes and peaches, but could not recall swallowing a pit. Because of the ever-present possibility of obstruction, laparotomy was suggested. For personal reasons the patient decided to postpone the operation and was discharged from the hospital on a bland diet.

Fig 1.—Scout film of abdomen showing pit.

He was readmitted May 5, 1948, for laparotomy. There had been no change in the symptoms since the previous admission. Physical examination revealed few abnormalities. Blood pressure was 140/70, pulse 72, temperature 98.6° F. The chest was emphysematous. A right direct inguinal hernia was present. Laboratory studies were within normal limits except for blood volume studies. These showed an estimated need of 500 c.c. to restore the calculated normal volume.

X-ray Studies.—

Gastrointestinal series: Preliminary film showed an oval density with a dense rim and scalloped border in the lower abdomen (Fig. 1). It was without essential change from that seen on previous x-ray examinations. The esophagus, stomach, and duodenal bulb showed no abnormality. The small bowel was studied with the aid of hourly films. The proximal part of the intestines (jejunum) appeared normal. The rounded body was seen in the ileum, but there was no distention of the ileum proximal to it. There were two stenotic areas in the

terminal ileum. There was no delay in the small intestine transit time. Seven-hour film showed the foreign body partly covered by barium; most of the barium was in the terminal ileum and distal colon. The appearance was that of regional ileitis with some points of stricture and with an intraluminal foreign body. This might have been a fruit pit. The strictured areas in the terminal ileum were approximately the same as those seen on Sept. 2, 1944.

Chest x-ray: The chest film was negative.

Course in the Hospital.—The patient's blood volume deficit was corrected with a transfusion of one pint of blood. A Miller-Abbott tube was inserted forty-eight hours prior to surgery. A low residue diet was ordered after this. The night before the operation the patient was given 2 gr. of Luminal Sodium and a soapsuds enema until the return was clear. Penicillin was utilized twenty-four hours before operation and continued for several days thereafter.

On May 13, 1948, a resection of the ileum and removal of the foreign body with an ileoileostomy and appendectomy was performed. The patient was prepared and draped in the usual manner. The abdomen was explored through a right transverse incision below the umbilicus. Approximately 45 cm. proximal from the ileocecal valve a stricture of the ileum was found. Immediately distal to this a Meckel's diverticulum 4 by 4 cm. was present. Proximal to the Meckel's diverticulum there was a moderate amount of dilatation of the remainder of the ileum. A foreign body was palpated within the proximal ileum. The ileum was skeletonized for a distance of about 12 cm. and the involved portion was removed between clamps. Both ends of the severed ileum were then closed in two layers, the first with catgut and the second with interrupted black silk sutures. A side-to-side ileoileostomy was then performed in two layers, utilizing catgut for the inner layer, and interrupted black silk for the outer. The mesentery was closed with silk sutures. An appendectomy was performed employing the ligation technique. The closure was in layers with black silk sutures, except for the peritoneum, which was closed with catgut. During the operation 500 c.c. of blood were given.

When the specimen was examined immediately after the completion of the operation, a stenotic ring, barely admitting the tip of the little finger, was found at the proximal junction of the diverticulum and the ileum. About one-third of the long diameter of the prune pit was sufficient to occlude the lumen. The pit could not be wedged into the stenotic area to produce a permanent occlusion.

The postoperative course was uneventful. Penicillin was given prophylactically. The patient was ambulatory on the first postoperative day. Codeine was given for one postoperative day to control pain. The Miller-Abbott tube was removed on the second postoperative day. During the interval in which the tube was in place the patient was supported with intravenous infusions of protein hydrolysates, glucose, and glucose and saline solutions. After the tube was removed the patient was placed on a progressive enterostomy diet. On the fifth postoperative day he was afebrile and remained so thereafter. On the seventh postoperative day the sutures were removed. The wound healed *per primam.* From the tenth postoperative day onward the patient was eating a regular diet.

Pathology Report.—

Gross description: The specimen consisted of a section of ileum and appendix. The section of ileum measured 2½ cm. in length. The specimen had been opened previously and appeared somewhat shrunken, however, the circumference of the bowel appeared smaller than average. The lumen of the diverticulum was empty and appeared lined by intestinal mucosa. No gross changes were noted. Presented separately was a pit which was elongated, with pointed poles. The surface of the pit was smooth and suggestive of being a prune pit. The appendix measured 5 cm. in length and 0.5 cm. in diameter. The serosa was smooth and glistening. Coronal sections showed the lumen to be patent to the tip, and filled with fecal material. The mucosa was intact and the muscular coat was of average width.

Microscopic description: Section 1 showed ileum. The mucosa showed somewhat of a polypoid proliferation with moderate edema of the mucosa and submucosa. The stroma contained increased numbers of eosinophiles and mononuclear cells. There was a superficial ulceration involving the mucosa. The edges of the ulcer were sharp and showed considerable inflam-

matory reaction. The base of the ulcer was formed by vascular granulation tissue, showing diffuse infiltration with polymorphonuclears and eosinophiles. The muscular coat and serosa showed no appreciable changes.

Section 2, taken from Meckel's diverticulum, showed the intact mucosa formed by intestinal mucosa. The two muscular layers were thin. Three further sections showed appendix. The lumen was filled with fecal material and was narrow. The mucosa was intact and showed marked hyperplasia of the lymphoid tissue, forming large lymph follicles with large distinct germ centers. The submucosa was thin and the muscular coat showed no appreciable changes.

Diagnosis.—Diagnosis was chronic and subacute ulcer of the ileum, Meckel's diverticulum, and lymphoid hyperplasia of the appendix.

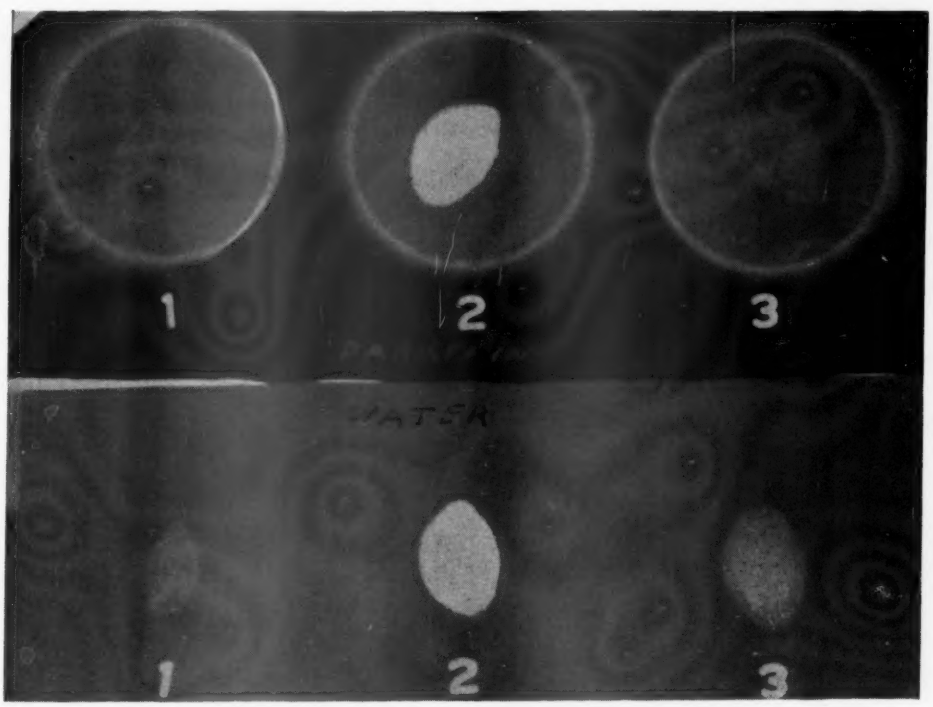

Fig. 2.—X-ray film of pits in paraffin and in water. No. 2 is the pit which was removed.

DISCUSSION

It is unusual for foreign bodies to remain in the intestinal tract more than a few days. In this instance, the stenosis at the proximal junction of the Meckel's diverticulum and the ileum probably prevented the fruit pit from passing. Fortunately the diameter of the stenosis was sufficiently small to prevent the pit from wedging into it, with consequent intestinal obstruction, and yet was large enough to permit free passage of intestinal contents. The etiology of the stricture is unknown, but its constancy may explain the previous x-ray diagnoses of regional ileitis.

When this case history was told to a consulting radiologist, he suggested that the pit had absorbed a large quantity of calcium salts during its residence. The possibility was investigated and demonstrated in several ways. Fig. 2 shows an x-ray view of the calcified pit[2] between two normal pits. A rim of

calcification was found when the pit was sawed in half. Comparison of the pit with prune and peach pits confirmed the diagnosis of prune pit made by the radiologist.*

CONCLUSION

A case has been presented in which a prune pit remained in the ileum for at least four and one-half years in association with a stricture at the site of Meckel's diverticulum.

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*Radiologist, Dr. Kirsh.

RUPTURE OF PYOGENIC HEPATIC ABSCESS INTO HEPATIC VEIN

Report of a Case

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THIS is a case report and short discussion of a death caused by a rarely reported mechanism.

This 28-year-old patient was struck across the abdomen by a railroad tie. He entered the Veterans Administration Hospital three days later because of persistent abdominal pain and distention. The abdomen, examined on admission, had minimal generalized rigidity and tenderness; a paracentesis disclosed free bile. A diagnosis of visceral rupture led to immediate laparotomy.

At surgery 2 liters of bile were aspirated. Careful exploration revealed a 1.5 cm. laceration that appeared to be in the quadrate lobe of the liver. It was inaccessible for suturing; Penrose drains were placed to the area and the abdomen closed. He was ambulatory the next day. Penicillin and streptomycin were given daily. By the fifth postoperative day the temperature was normal, and the streptomycin was discontinued. Profuse bile drainage persisted, but otherwise he felt well.

On the tenth postoperative day he had a transitory episode of epigastric pain. Roentgenograms were taken of the chest and abdomen; they were interpreted as negative. Late that afternoon the epigastric pain recurred in agonizing degree. It radiated through to the back, lasted two hours and left him feeling "very bad." We could not assign a cause for the symptoms. When seen later he was sleeping quietly.

Next morning a catastrophic change in his condition was apparent. He was lethargic; temperature was 101.4° F., and white blood count was 24,500 with 98 per cent neutrophiles. Blood pressure was 70/40 mm. Hg. The presumption was that an intra-abdominal abscess had ruptured, causing an overwhelming toxemia; surgical exploration seemed indicated.

The upper portion of the abdominal cavity was explored; no entity to explain the toxemia was found. The hepatic laceration had become sealed. Blood given during surgery increased the systolic pressure.

Following surgery the pressure fell again to 70. A chest roentgenogram and an electrocardiogram were taken and found essentially normal. A blood culture was obtained, after which 1,000,000 units of penicillin and 1.0 Gm. of streptomycin were given intravenously. Penicillin, 200,000 units, was given every two hours, and 0.5 Gm. streptomycin was given every six hours thereafter.

The persistent shock was thought due to profound toxemia. That the ultimate outcome in such cases of toxemia is not affected by transfusions has been recognized clinically; this recently has been verified experimentally.[1,2] Because of this, and because an oliguria (attributable to the hypotension) was present, combating the shock with excessive parenteral fluids seemed injudicious. However, 1,500 c.c. of blood were given through the day (loss during surgery was negligible). Neosynephrin was administered intravenously in divided doses; only a transient pressure elevation resulted. Parenteral intake this day was 3,500 c.c.; output was 175 c.c. The temperature rose to 104° F.

The next day Neosynephrin in 5 per cent glucose solution was slowly infused intravenously for several hours; the systolic response was slight, the blood pressure remaining about 70. Moist râles were audible throughout the lungs. The parenteral intake this day was 1,750 c.c. and urinary output 250 c.c. His condition continued critical.

The next day the blood pressure ranged between 100 and 114. He improved thoughout this day, was rational, and rested quietly. Temperature spiked between 99 and 103° F. He received 1,000,000 units of penicillin intravenously twice this day in addition to the regular

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penicillin dosage. Parenteral intake this day was 1,500 c.c., urinary output 80 c.c. Hemoglobin was 14 gm., white blood count 16,500, and blood urea nitrogen 90.

He died suddenly during the early morning of the third postoperative day. The blood culture previously obtained yielded a heavy growth of *Proteus vulgaris.*

An autopsy was performed three hours after death. Significant observations were limited to the abdomen. A thin layer of fibrinopurulent exudate held the liver and adjacent bowel together. The gall bladder, extrahepatic biliary ducts, hepatic artery, and portal vein were normal. The posterior portion of the right lobe adjacent to the quadrate lobe presented a hemorrhagic, friable area containing a laceration 1.5 cm. in length. On section, a thin band of hemorrhagic necrotic material extended from the laceration to an abscess cavity 4.5 cm. in diameter situated near the superior surface of the right lobe (see Fig. 1). The cavity was filled with a hemorrhagic, necrotic, putrid material. The cavity walls were well demarcated and appeared a dark greenish-black; the cavity communicated directly with

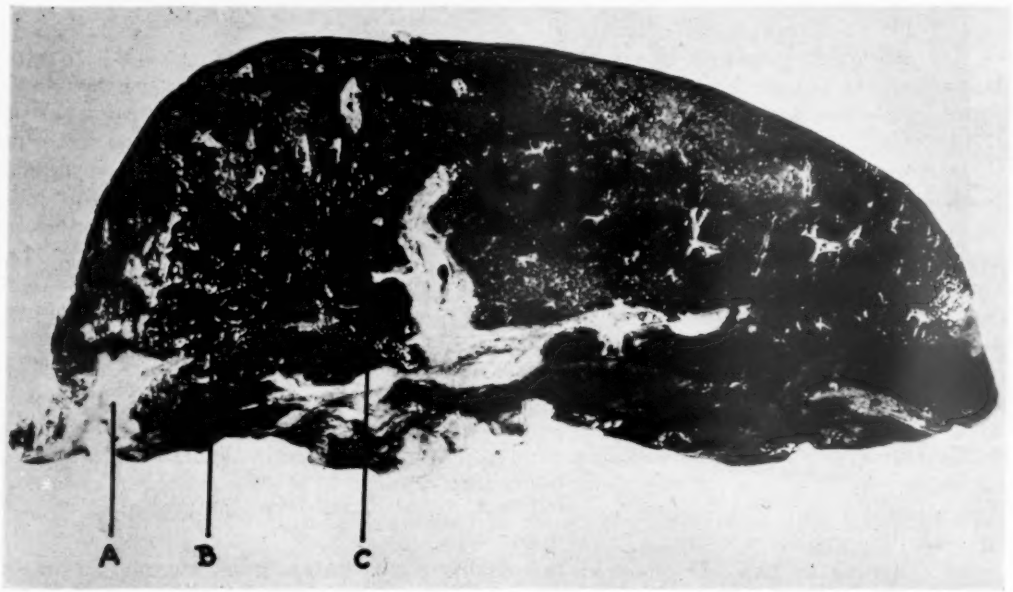

Fig. 1.—Sagittal section through medial portion of the right lobe of the liver. *A*, Hepatic radicle (thrombus has been removed to illustrate better the vessel). *B*, Abscess cavity. *C*, Band of hemorrhagic necrotic material extending from area of laceration to the abscess cavity.

a large radicle of the hepatic vein. A friable thrombus extended from the abscess cavity along the vein to its junction with the inferior vena cava. Microscopic examination of the abscess wall revealed an acute purulent exudate and necrotic debris, surrounded by a thin band of granulation tissue. The parenchyma at a distance from the abscess cavity had gross and histologic evidence of mild passive congestion.

Material from the liver abscess and a post-mortem blood culture yielded very heavy growths of pure *P. vulgaris* that was not sensitive to streptomycin in vitro.

DISCUSSION

Circumscribed trauma to the abdominal wall is liable to injure the intestine or kidney; force applied diffusely (as in this case) is more likely to injure the liver, spleen, pancreas, or blood vessels.[1] Fortunately, liver ruptures usually occur along the central plane, where large blood vessels and bile ducts are absent.[2] Following trauma the pulpified hemorrhagic area in the liver may be absorbed or it may become infected and develop into abscesses.[3, 4] The vascularity

and extensive reticuloendothelial system of the liver ordinarily forestalls this, and abscess following external nonpenetrating trauma is rare.[3, 5, 6] When it does supervene, it is usually single.

Pain occurs with hepatic abscess in about 80 per cent of cases; it is usually constant and without relation to food and severest when the abscess is in the upper surface of the liver.[7] The classic "picket fence" fever and recurrent chills rarely occur with a solitary abscess. Hepatomegaly develops only if the abscess is large.[8] Jaundice is a late and serious sign; it may be produced by direct pressure on large biliary ducts.[6] Nausea and vomiting rarely occur. This has diagnostic value, for most other conditions that cause pain in the right upper quadrant, plus chills and fever, have associated nausea and vomiting.[6] However, diagnosis of a solitary liver abscess is difficult.

The organisms that cause pyogenic abscesses[5] are listed in Table I.

TABLE I

ORGANISM	PER CENT
Bacillus coli	31.0
Streptococci	20.0
Staphylococci	17.2
Combination of these	17.2
Total	95.4

Mycotic abscesses are uncommon and when encountered are ordinarily due to Actinomyces.[9] Rarely *Clostridium perfringens, Pseudomonas pyocyanea, Eberthella typhosa,* leptothrix, streptothrix, salmonella, spirochetes and gonococci cause liver abscesses.[8] There is a mixed infection in 15 to 20 per cent of nonoperated amoebic liver abscesses.[10]

If drainage of a pyogenic abscess is not done, the mortality is very high. In a collected series of cases of 102 patients not operated upon, the mortality was 100 per cent. The affect of antibiotic therapy in altering this mortality remains to be seen. It is noteworthy that this patient received continual antibiotic therapy from time of admission. Extension of the abscess into one of the adjacent viscera, or more rarely thrombosis and embolism, is the commonest complication. Abscesses most frequently involve the pleura, lung, peritoneum, and pericardium, usually by direct extension.[5] The stomach, colon, lumbar region, bile ducts, inferior vena cava, and kidney follow in order, with infrequent cases being reported.[11] McKnight (1928) found only twelve cases of hepatic abscess with rupture into the hepatic vein or vena cava; most of these were amoebic abscesses.

In this patient a pulpified area deep in the right liver lobe became infected, and an abscess formed. There were no symptoms such as pain, fever, or jaundice to suggest this until the vein wall was eroded. Then, a massive influx into the blood stream of necrotic material heavily contaminated with *P. vulgaris* occurred. Although the preoperative diagnosis was a ruptured intraperitoneal abscess, no external evidence of the liver abscess was found during surgery. A hepatic abscess developing after nonpenetrating trauma and rupturing into the hepatic vein (the organism being *P. vulgaris*), serves to make this case unique.

In retrospect, the exact diagnosis might have been surmised from the history of liver injury and the sudden severe toxemia and collapse thirteen days later.

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ABDOMINAL APOPLEXY

Report of a Case

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ABDOMINAL apoplexy, or acute spontaneous intra-abdominal hemorrhage, is rarely considered in the differential diagnosis of acute conditions of the abdomen. The infrequency of its consideration is borne out by the fact that in the thirty-five cases thus far reported in the literature the diagnosis of abdominal apoplexy was made preoperatively in only one, reported by Crile.[1]

The earliest report is generally credited to Barber,[2] in 1909, although a critical review of the paper leaves its priority in serious question, since the patient was a newly parturient woman who was believed to be bleeding from a vessel in the pelvis. The bleeding vessel was not identified, and there is no certainty that the hemorrhage was not secondary to the trauma incident to delivery.

During the next three decades, reports were scanty. Green and Powers,[3] reporting in 1931, were able to find only four cases other than Barber's, three of them well-documented and the fourth incompletely reported and quite questionable. Two further papers were published prior to 1935, when Thompson and Dunphy[4] reviewed seven cases from the literature and added their own case.

Since 1935, due either to an increased awareness of the syndrome or to better reporting of cases, the number has increased more rapidly. A total of twenty-one reports had appeared by 1941[5]; and in 1947, Tanna,[6] in an excellent review of the literature, listed thirty-two cases and added three more.

It is our purpose to add a thirty-sixth case from the records of the Mary Imogene Bassett Hospital in Cooperstown, N. Y.

CASE REPORT

D. K., a 73-year-old, single, white man, a farmer, was admitted to the hospital complaining of severe lower abdominal pain of five hours' duration.

Family History.—The family history was noncontributory.

Past History.—The patient's health had been generally good until the preceding two or three years. He had suffered from headaches of mild type for thirty years, which had been relieved by Aspirin. He had noted mild dyspnea for two or three years, with rare paroxysmal, nocturnal dyspnea, and had experienced palpitation for twelve months prior to entry. Ten and four months previously he had had episodes of cardiac failure with increased dyspnea and edema to the knees. He had been digitalized each time, with prompt relief, and at the time of admission was being maintained on 0.2 Gm. of digitalis leaf daily. For about six months the patient had complained of occasional dull lower abdominal pain unrelated to food. There was no history of intolerance to any food nor of nausea. The patient's bowel habits were irregular, but there had been no recent change. Nocturia, occurring once or twice nightly, had been present for five years. There had been, lately, a decrease in memory for recent events, and mild insomnia, and he was said to have become more irritable in the recent past.

Illness at Time of Admission.—Five hours prior to admission, while sitting quietly at home, the patient was seized with sudden, severe, stabbing pain in the lower abdomen. He had previously felt well and had completed his morning chores about the farm. The pain was nonradiating, and it was not affected by postural change. It increased to a peak in about twenty minutes and was sustained at that level. There was no nausea, vomiting, diarrhea,

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or urinary distress. The patient was seen by his doctor, who administered 0.015 Gm. of morphine without immediate relief of the pain, and he was subsequently admitted to the hospital by ambulance.

Physical Examination.—The patient's temperature on admission was 99.4° F.; pulse rate 88; respirations 18; blood pressure 185/95. He lay quietly in bed in no acute distress and appeared somewhat lethargic. Cooperation was poor. There was marked cyanosis of the lips and nail beds. The pupils were contracted, but reacted to light and accommodation. The patient was edentulous except for a few carious stumps, and the buccal mucosa was pale. Examination of the chest revealed that the lateral cardiac border was 13 cm. from the midsternal line, and there was a harsh apical systolic murmur. The lungs were normal. The abdomen was rigid and generally tender, with the greatest tenderness located just above the umbilicus. No rebound tenderness could be elicited, and deep palpation was precluded by spasm. There was no distention, and no free fluid could be made out. Rectal examination revealed a moderately enlarged prostate, but no tenderness. The neurological examination was negative.

Laboratory studies showed a hemoglobin of 10.7 Gm., a leucocyte count of 8,600 with 84 per cent polymorphonuclear leucocytes, and a serum amylase of 19.3 Myers-Free units. An electrocardiogram showed digitalis effect and acute myocardial damage without infarction. A roentgenogram of the abdomen in the erect position showed no gas under the diaphragm, no distended coils of intestine, and no calculi.

It was felt that virtually all causes of acute abdominal pain had to be considered, but that the most likely diagnoses were acute pancreatitis, mesenteric thrombosis, or atypical appendicitis as it so often appears in the aged.

Hospital Course.—In view of his good general condition, and since there was no increase in symptoms, it was decided to treat the patient conservatively and await developments. Penicillin was administered, and he was given intravenous fluids. During the night, the tenderness appeared to be localizing in the right lower quadrant, and the leucocyte count rose to 10,300. The following morning, eighteen hours after admission, an exploratory celiotomy was done through a McBurney incision under nitrous oxide-oxygen-ether anesthesia, with a preoperative diagnosis of acute appendicitis.

On incising the peritoneum, a large amount of bright red fluid blood escaped, followed by a moderate amount of clotted blood. Underlying the cecum was a large hematoma, and on further exploration a bleeding branch of the right colic artery was identified and ligated. The abdomen was closed without drainage.

The postoperative course was uneventful except for a mild urinary retention and some nocturnal disorientation. The patient was discharged, without symptoms, to his local doctor on the fifth postoperative day. He was seen in the outpatient department ten days later, and at that time the wound was healing well, and he had no complaints. He failed to return again to the follow-up clinic, but his doctor reported, eighteen months later, that he had had no further abdominal difficulty.

Comment.—Abdominal apoplexy, like the cerebrovascular type, occurs in elderly, hypertensive, arteriosclerotic individuals; the mean age in Tanna's series was found to be 53, with men exceeding women in a ratio of 2.5 to 1. The bleeding can occur from any vessel within the abdominal cavity. The onset is sudden, and the pain is described as being of two types. If the bleeding is confined to the mesentery, the pain is dull and sickening in character, due to stretching of the peritoneum of the mesentery, and is similar to that incident to traction on the bowel. If the bleeding occurs directly into the free peritoneal cavity, or if the mesenteric hematoma ruptures into the peritoneal cavity, the pain is severe, sharp, and prostrating, resembling that of ruptured ulcer or acute pancreatitis.

The abdominal signs of intraperitoneal hemorrhage are ordinarily those of acute, diffuse peritoneal irritation, with no well-defined localization. Accompanying these local manifestations are the general signs of acute hemorrhage,

with a decrease in hemoglobin and erythrocyte count; the leucocyte count is usually increased. Shock may be present or absent, depending on the severity of the blood loss.

It is probable that minor intra-abdominal hemorrhages from small vessels may subside without operative procedure. In general, however, as soon as the diagnosis of abdominal apoplexy is made, the treatment of choice is surgical. Shock, if present, should be adequately treated before operation is undertaken. If, in spite of treatment, shock is still present, abdominal operation should nevertheless be seriously considered, for if the bleeding is continuous it may not be possible to improve markedly the patient's condition until the hemorrhage has been stopped. Transfusion should be under way at the time the operation is started and preparations made to continue it in large volume as long as is necessary. It seems reasonable to believe that lives might be saved by this energetic therapy, as has been proved in certain cases of abdominal war wounds complicated by hemorrhage (Snyder[7] and others).

From the point of view of their cardiovascular system, patients with abdominal apoplexy are often poor surgical risks. Careful consideration should be given to the preoperative medication required, including digitalization where indicated, and to the most desirable type of anesthesia for the individual patient.

The increasing frequency of reports of abdominal apoplexy in the last ten years, and particularly reports of more than one case by the same author, lead one to suspect that this condition is by no means as rare as might be assumed from the total number of cases reported.

It is clear that, particularly in the group of elderly, hypertensive, sclerotic patients, this diagnosis should be considered in evaluating acute abdominal conditions.

SUMMARY

1. The literature reporting thirty-five cases of abdominal apoplexy is briefly reviewed.

2. The disease may mimic a number of acute abdominal conditions, and the correct preoperative diagnosis of abdominal apoplexy has been made only once. It should be suspected in connection with any acute abdominal condition occurring in an elderly, sclerotic, hypertensive patient.

3. A case is presented of abdominal apoplexy due to rupture of a branch of the right colic artery in a 73-year-old man. The patient was operated upon and the bleeding vessel ligated. Recovery was uneventful.

4. The importance is stressed of adequate preoperative measures, including the treatment of shock with massive transfusions when indicated.

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PANCREATIC CALCULOSIS FOLLOWING TRAUMA

Report of a Case

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THE increasing clinical significance of pancreatic calculosis is well attested by the number of reviews of the subject and case reports published, particularly in the past decade. Comprehensive reviews of the literature by Haggard and Kirtley[1] in 1939, King and Waghelstein[2] in 1942, and by Lionello, Ficarra, and Ryan[3] in 1944 obviate the necessity for a review at this time. Still more recently, Martin and Canseco[4] have presented a review of twenty-one cases at the Johns Hopkins Hospital with an excellent description of the clinical pictures, pathologic changes, and varied types of treatment of this relatively rare condition, and Whipple[5] has recently described his indications for pancreatectomy in these cases.

The exact cause of pancreatic calcification apparently is not known. Ackman and Ross[6] believed that most of the accumulated evidence points to a primary inflammatory condition with stasis as a secondary factor. This is borne out by accompanying pathologic changes in the pancreas and by the presence of calcium in these calculi which is absent in normal pancreatic secretion. However, Martin and Canseco stated that sections made from biopsy and autopsy specimens in their series gave no consistent evidence of an underlying infectious basis. Cases of pancreatic calculosis have not been consistently associated with biliary tract disease or chronic alcoholism, but these have been mentioned as contributing factors. The length of time between the onset of a postulated previous pancreatitis and the appearance of calcification is also not known.

This report presents a case of pancreatic calculosis of the body and tail of the pancreas, treated by partial pancreatic resection, in which the origin of the calculi was related to a nonpenetrating abdominal injury. Although it is well known that traumatic pancreatitis may follow abdominal injury,[7, 8] and that such injuries are sometimes complicated by hematoma, abscess, or pseudocyst formation, a review of the literature fails to reveal any case in which trauma to the pancreas has resulted in calculus formation.

CASE HISTORY

D. W. W. (Hospital No. 159-819), a 37-year-old white man, was admitted to the medical service of the Wadsworth General Hospital on Jan. 26, 1948, complaining of recurrent episodes of severe abdominal pain of five years' duration. He had been in excellent health until February, 1943, when he was struck in the left flank by a rifle butt. One month following this injury he suddenly and spontaneously collapsed and was taken to a military hospital, where an emergency laparotomy was performed and a ruptured

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spleen was found and removed. Since that time he had had recurrent severe attacks of left upper quadrant pain occurring at variable intervals, lasting from several hours to five or six days, and frequently requiring opiates and hospitalization for relief.

In February, 1947, he was admitted to another hospital with a recurrence of severe left upper quadrant pain. An x-ray examination of the abdomen revealed some calcifications in the left upper quadrant which were interpreted as being located in the body and tail of the pancreas. A laparotomy was performed on March 28, 1947, and an old hematoma containing calcific particles and fragments of splenic pulp was removed from the region of the greater curvature of the stomach and the left subphrenic region. The pancreas apparently was not explored at this operation. The patient had several recurrent attacks following this surgery, during one of which an elevated serum amylase of 1,023 units was found for the first time, but the patient was put on a low fat diet, and discharged with no further treatment.

Fig. 1.—Anteroposterior view showing calcifications in region of tail of pancreas.

On admission to this hospital about eight months following the aforementioned operation, the patient complained of recurring attacks of greater frequency and increasing severity, with radiation of the pain to the middorsal region. During such attacks of several days' duration the stools tended to become pale, frothy, and bulky. There had been no loss of weight or strength and no discomfort between attacks of pain. There was no history of alcoholism or relationship of the attacks to the intake of food. Physical examination was essentially unrevealing except for a healed left subcostal scar, a liver edge which was palpable just at the right costal margin, and slight diffuse tenderness in the left upper quadrant.

Routine urinalysis, complete blood count, serology, and sedimentation rate were normal. Liver function tests were all within normal limits. The serum amylase during several attacks of pain ranged from 236 to 1,110 units. Blood lipase activity on one occasion was equivalent to 2.6 c.c. of 1/20 N NaOH. Blood calcium and phosphorus levels were normal. A glucose tolerance test revealed a slightly elevated curve. A flat film of the abdomen revealed calcifications in the region of the tail of the pancreas (Figs. 1 and 2). Gall bladder studies showed a normally functioning gall bladder, and an upper gastrointestinal study revealed a normal stomach and duodenum.

After admission, the patient continued to have frequent severe attacks of epigastric and left upper quadrant pain which at first could be readily relieved by the intramuscular administration of 500 mg. of tetraethylammonium chloride (Etamon chloride). After a number of attacks, however, Etamon chloride in doses of 1,000 mg. produced less effective results and opiates were required for relief.

The patient was seen in consultation on Feb. 14, 1948, at which time resection of the involved portion of the pancreas was advised. Operation was performed on Feb. 26, 1948, under continuous spinal anesthesia. The pancreas, exposed through the gastrocolic ligament, was found to be narrow and firm throughout and somewhat dark reddish in color. There was marked atrophy and fibrosis involving most of the body and tail of the pancreas to the left of the superior mesenteric vessels, and the pancreas was bound down in its bed posteriorly by a diffuse peripancreatitis. Numerous small stones could be palpated in the tail of the pancreas, imparting a gritty sensation. The head of the pancreas was

Fig. 2.—Lateral view showing calcification.

somewhat smaller and firmer than normal but no stones could be palpated within it, and the atrophic and fibrotic reaction was much less marked than that of the body and tail. The gall bladder was normal, and the common duct appeared slightly dilated but was patent into the duodenum. A resection of the body and tail of the pancreas and a choledochostomy were performed. Photographs of the intact and bisected specimen showing the intraductal calculi are shown in Figs. 3 and 4. The pathologist reported a chronic fibrosing interstitial pancreatitis with intraductal calculosis.

The patient obtained dramatic relief from the almost daily attacks of pain which he had been having up to the day of surgery. The postoperative course was entirely uneventful, and the T tube was removed from the common duct after six weeks. The patient was discharged on April 8, 1948, gaining weight and completely free of pain for the first time in five years.

He returned to the hospital a few months later because of right upper quadrant pain which radiated through to the back in the region of the inferior angle of the scapula, and stated that he had been having pale stools which seemed "greasy and fatty," and there

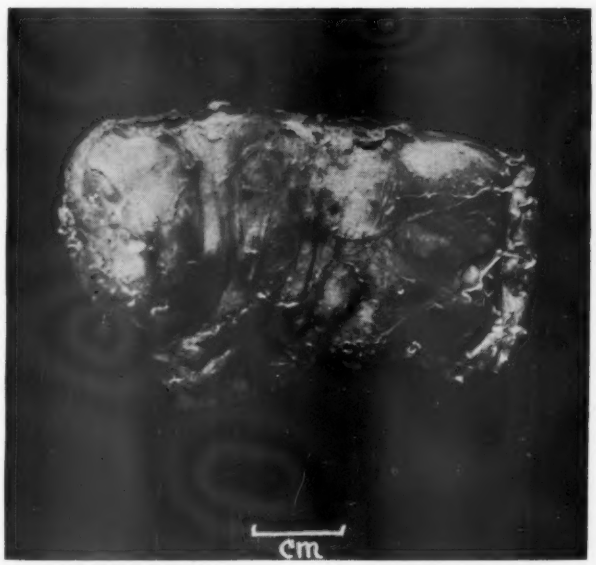

Fig. 3.—Intact specimen.

Fig. 4.—Bisected specimen showing intraductal calculi.

was a weight loss of about ten pounds. Serum amylase and lipase studies done on several occasions during episodes of right upper quadrant pain were within normal limits. Stool examinations revealed increased free fat and fatty acids, and duodenal drainage revealed

decreased pancreatic enzyme activity. These findings, plus a mild diabetic glucose tolerance curve, indicated a pancreatic insufficiency of moderate degree. The patient responded nicely to a high protein, high carbohydrate, low fat diet, and 15 units of regular insulin daily; the fatty, frequent stools were completely controlled by the use of pancreatin and also Tween 80, as recommended by Jones and associates.[9] He gained twelve pounds in weight on this regime, but occasional episodes of mild right upper quadrant pain continued. A gall bladder study at the time of this admission showed a nonfunctioning gall bladder, and a gastrointestinal series suggested an extrinsic mass producing some anterior displacement of the distal one-third of the stomach, indicating the possibility of a pancreatic cyst. However, the symptoms were not severe enough to warrant further surgical intervention at this time and he was again discharged on Sept. 8, 1948.

SUMMARY

A case is presented of calculosis of the body and tail of the pancreas treated by subtotal pancreatic resection. The onset of this condition was preceded by a nonpenetrating injury of the abdomen severe enough to have caused a delayed splenic rupture. Following injury, the patient suffered recurrent attacks of acute pancreatitis, pancreatic calculus formation, and evidence of progressive pancreatic insufficiency. No previous reference in the literature has been found in which the origin of pancreatic calculi was so clearly related to abdominal trauma.

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PANCREATIC CYSTGASTROSTOMY

Discussion and Report of Case

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PANCREATIC pseudocyst has been accorded increasing interest in recent years because of the development of several methods of operative attack. The disease is sufficiently rare so that no adequate series of the several methods of internal drainage has been collected for evaluation. Therefore, the contribution of another case report to the literature is deemed worth while.

Pancreatic cysts were classified by Robson and Moynihan[16] in 1903, then reclassified by Mahorner and Mattson[10] in 1931, in whose paper is an excellent discussion of the pathogenesis of the several types of cyst. Clinical reviews of the subject are found in several papers which emphasize the physical and symptomatic diagnosis. Prognosis and the complication of perforation are discussed by Brunschwig,[2] and by Koucky, Beck, and Todd.[9]

Modern treatment of pancreatic cyst is surgical. Three general possibilities are recognized. Total excision of the cyst is seldom possible because of the extent of the adhesions of the cyst to neighboring viscera and to important structures such as the middle colic vessels. When the cyst is small enough to be separable and has a pedicle narrow enough to be resectable, along with the tail of the pancreas and spleen if necessary, total excision offers primary healing and uncomplicated convalescence, and is the treatment of choice. More often, however, because of the situation of the cyst or the general condition of the patient, a simpler procedure is necessary.

Gussenbauer,[2] in 1883, originated the treatment of pancreatic cyst by marsupialization. This procedure has since been the standard one, and has produced consistently satisfactory results with low mortality. Disadvantage of the method lies in the prolonged drainage from the cyst before the latter becomes obliterated with granulation tissue. This discharge lasts from a few days to months or even years in individual cases, and may be extremely irritating to the skin. Meyer[11] has reported cases of sepsis, of ruptured cyst, of fecal fistula, and of incomplete drainage as complications following marsupialization.

Principally to avoid the chronically draining sinus, the third plan of surgical attack has been developed. Occasional rare cases of spontaneous internal drainage have been reported (cited by Brunschwig[2]), and it was perhaps from these that the idea of constructing a cyst-visceral fistula came. Jedlicka[11] was the first to advise internal drainage, and presented a successful case of anastomosis to the stomach. Jurasez[11] soon reported another case. Cystgastrostomy is also recommended by Harries.[6] Kerschner[8] reported two cases of successful cystduodenostomy. Cystjejunostomy was reported by Hahn[11] and Chesterman.[3] Adams and Nishijima[1] added a short-circuiting jejunojejunostomy to the procedure, while Migliaccio and Laurelli[12] went further and applied the Roux Y principle. Cystcholecystostomy has received considerable attention (Hames-

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fahar, Walzel, Neuffer, Meyer[11]), with emphasis on the utilization of the absorptive powers of the gall bladder.

Certain authors have expressed preference for particular operations and have proposed interesting arguments and facts to support their opinions. The best procedure for internal drainage will probably not be identified until further cases can be collected. Following is the report of a case of pancreatic cyst treated by cystgastrostomy and complicated by peptic ulceration of the stomach with repeated massive hemorrhages. This case would suggest that cystgastrostomy might be a procedure to avoid.

CASE REPORT

L. E., a 38-year-old white salesman, was admitted to St. Mary's Hospital Nov. 5, 1944, with acute severe midepigastric pain. He had a leucocyte count of 27,000, and serum amylase was 900 units. Under nonoperative management he gradually improved and was discharged after fourteen days. The diagnosis was acute pancreatitis.

The patient did poorly after discharge and was readmitted to the hospital three months later, having lost fifty-two pounds in weight. He was suffering from severe abdominal pain with vomiting, and was bearing a large, firm mass which filled the left upper quadrant of the abdomen. After a week of preoperative preparation, an exploratory laparotomy for pancreatic cyst was carried out. A cyst 5 inches in diameter was found, presenting between the stomach and colon, and causing irritation and inflammation of the gastrocolic ligament and gangrene of part of the omentum. Ascitic fluid (7½ quarts) was suctioned from the abdominal cavity. The cyst wall was 1 cm. in thickness and densely adherent to the posterior gastric wall. Open anastomosis between the cyst and the greater curvature of the stomach was carried out, plus cholecystostomy with removal of gallstones.

The patient did well following surgery until the sixth day, when he passed gross blood by rectum and by emesis and went into shock. Treatment with blood infusion and other supportive measures brought gradual response. Four more episodes of massive hemorrhage with shock occurred in the next twenty-eight days, and reoperation was deemed necessary.

On the forty-fifth postoperative day the patient was re-explored. The pancreatic cyst had virtually disappeared, with only a residual thick scar on the greater curvature of the stomach. Anterior gastrotomy revealed a linear ulcer on the posterior gastric wall adherent to the pancreas, presumably the source of the bleeding. Simple excision of the ulcer with dissection of the stomach free from the pancreas, followed by inversion and closure of the gastric wall, was selected as the least formidable and most suitable procedure, despite the recognized possibility of recurrence.

The patient again suffered massive gastrointestinal hemorrhages with almost fatal shock on the seventh, ninth, and sixteenth postoperative days. In the absence of patent superficial veins, sternal transfusion became a lifesaving expedient. Because of the likelihood of further hemorrhages, on the twentieth day the patient was returned to surgery for a subtotal gastric resection. The lesser omental sac was obliterated with dense gastropancreatic adhesions. No cleavage plane was identifiable and dissection was bloody and almost impossible. Subtotal resection was possible only with antral mucosectomy and an antecolic gastrojejunostomy. A Witzel enterostomy for subsequent feeding was also performed. A peptic ulcer was found at the site of the old cystgastrostomy. Bleeding from the dissection about the ulcer bed was extremely heavy and was controlled with difficulty with several pairs of forceps which were left in place, their handles taking exit through the wound after closure. Liver biopsy showed severe hepatic degeneration and periportal exudate (acute hepatitis). The patient was returned to bed after the administration of 11,000 c.c. of blood during six hours of surgery.

On the first postoperative day the patient's temperature rose to 105.2° F., pulse to 180. On the second day deep icterus and bilirubinuria appeared. Despite all this the patient took 3,000 calories per day via the enterostomy tube and began to improve. The

forceps were removed at nine and ten days without mishap. The wound healed well, and the patient was discharged after 120 hospital days. In April, 1948, he had regained his weight, had a competent abdominal wall, and was earning his living.

DISCUSSION

There is no positive evidence that the pancreatic cystgastrostomy was the cause of the peptic ulcer in this patient. However, in a patient 38 years old with no previous history of ulcer, the onset of severe peptic ulcer with hemorrhage six days after cystgastrostomy seems to justify the assumption of causation or precipitation. That the ulcer was not a product of pancreatic digestion is suggested by the completed disappearance of the cyst by the time of the second operation, when hemorrhages were still occurring. Unfortunately, no enzyme studies of the cyst fluid were done. Pathologic report was acute benign ulcer.

SUMMARY

The presented case of pancreatic cyst is unusually interesting in the following particulars.

1. The development of the cyst occurred under observation following an observed attack of acute pancreatitis.

2. The patient survived a six-month period of severe illness including severe malnutrition, three major operations, eight episodes of profound hemorrhagic shock, and acute severe hepatitis.

3. There were two supportive procedures whose value at particular times was critical: (a) blood transfusion to the extent of 60 pints of blood in 120 days, and (b) Witzel jejunostomy which allowed high-caloric alimentation at a time when oral feeding was impossible and liver depletion was imminent.

4. Peptic ulceration followed pancreatic cystgastrostomy so closely, with such catastrophic sequelae in this case, that we view this procedure with suspicion.

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PROGNOSIS OF SPLENECTOMY IN IDIOPATHIC THROMBOCYTOPENIC PURPURA ON BASIS OF MARROW STUDIES

Case Report

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IDIOPATHIC thrombocytopenic purpura is a clinical syndrome of unknown etiology manifested by a bleeding tendency. This bleeding tendency, when present in the course of the disease, gives rise to petechiae and purpura in the skin and mucous membranes. There may also be bleeding from any site of the body (epistaxis, hematemesis, melena, hematuria, menorrhagia, hemarthrosis, retinal and cerebral hemorrhage, and so on).

The disease is of two clinical types: the acute type is represented by a fulminating onset of this bleeding tendency which is sometimes fatal due to a cerebral-vascular hemorrhage or excessive blood loss; the chronic type is characterized by spontaneous relapses and remissions of the bleeding tendency.

Physical examination is entirely normal except for the purpura and other manifestations of the bleeding tendency.

The diagnosis is confirmed by certain laboratory findings, among which are: (1) decrease in the platelets of the peripheral blood, (2) prolongation of the clot retraction time, (3) prolongation of the bleeding time (coagulation time is normal), (4) positive Rumpel-Leede tourniquet test, and (5) microscopic study of the bone marrow.

All workers agree with the aforementioned data, but any further discussion of the disease is marked by many controversial theories which attempt to explain the phenomenon manifested.

It is generally agreed that one of the functions of platelets is to affect clot syneresis; thus, a prolonged clot retraction time is a factor in causing an increased bleeding time. It is postulated that bleeding persists because firm thrombi are not formed, and poorly retracted clots are easily dislodged from open ends of cut vessels.

The explanation for prolonged clot retraction and bleeding times is thus fairly well satisfied by thrombocytopenia per se, but a decrease in platelets cannot always explain the purpura and positive tourniquet test. During the course of chronic idiopathic thrombocytopenic purpura, the bleeding tendency usually appears and disappears with corresponding fluctuations in the platelet count. But in some instances, purpura and positive tourniquet tests have been observed to come and go without correlating decreases and increases in the number of circulating platelets. In attempting to explain this phenomenon, various authors have pointed to abnormal capillaries as the answer. Many

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claim that capillaries do not contract in thrombocytopenic purpura, and thus give rise to a bleeding tendency. Others postulate that one of the functions of platelets is the plugging up of normal fenestrae in capillary endothelium. This theory may state that a dearth of platelets causes bleeding if capillary holes are not plugged sufficiently or, conversely, that an increase in the openings of capillary endothelium gives rise to a decrease in platelets because so many platelets are utilized in blocking the capillary fenestrae.

Recently, however, Allen and associates[1] cited evidence that there may be an increased amount of a heparinlike substance in the blood of patients with thrombocytopenic purpura. It is thought that in some unexplained manner this substance may be a factor in causing the bleeding tendency. They have shown that when protamine sulfate or toluidine blue is administered intravenously to patients with thrombocytopenic purpura, there is an apparent cessation of purpura with the development of a negative tourniquet test. This change occurs without any alterations in the bleeding time, clot retraction time, or platelet level. It is therefore suggested that toluidine blue and protamine sulfate inhibit this heparinlike substance, and thereby decrease the purpuric tendency.

It should be realized at this point that the exact mechanism of the capillary factor is still unknown, and, as will be seen, the cause of the thrombocytopenia is also still unknown. However, the spleen must exert some effect upon the capillary factor and the number of circulating paltelets, because splenectomy cures most patients. A review of the recent literature reveals that there are two entirely different theories which attempt to explain the occurrence of thrombocytopenia on the basis of a splenic dysfunction.

Dameshek and Miller[2] published evidence that the spleen, by some unknown mechanism (''hypersplenism''), inhibits the megakaryocytes of the bone marrow in their production of platelets and their subsequent delivery of the platelets into the blood stream. Their cases of idiopathic thrombocytopenic purpura showed more megakaryocytes in aspirated sternal marrow than are usually present. However, platelet production by the megakaryocytes was markedly decreased, and there was a slight increase in the degenerative forms of megakaryocytes. Following splenectomy, this abnormal picture was changed to one of normal platelet production, with a simultaneous increase in the number of blood platelets. It is further stated that ''histologically the spleen shows little, if any, deviation from the normal''; these authors doubt that destruction of platelets by the spleen is much of a factor in producing thrombocytopenia. Valentine's[3] studies of the marrow in three cases of idiopathic thrombocytopenic purpura confirmed these findings. Hertzog[4] was also in agreement; his review of thirty-six autopsy cases revealed ''that the spleen shows no gross or microscopic changes which are characteristic of thrombocytopenic purpura.''

On the other hand, Doan and co-workers[5, 6, 7] uphold the belief that the decrease in the number of platelets in idiopathic thrombocytopenic purpura is due to their excessive destruction by the spleen. In their cases, supravital scrapings of freshly removed spleen have shown excessive reticuloendothelial hyperplasia and hyperphagocytosis of platelets. In addition, platelet counts of

the splenic artery have been observed to be significantly higher than those of the splenic vein. The administration of adrenalin is stated to increase the number of venous platelets, presumably by affecting a splenic contraction with expulsion of platelets into the splenic vein. This is the mechanism of the "adrenalin biopsy." Furthermore, these spleens show an excessive lecithinase activity as compared with the normal. These authors' observations of living bone marrow preparations have revealed that the megakaryocytes show no qualitative defects; they appear entirely normal, and are present in normal or slightly increased numbers.

If one is in accord with Dameshek's views, then it must be postulated that recurrences of thrombocytopenia after splenectomy must be due to either unremoved accessory splenic tissue or irreversible changes produced by hypersplenism. Doan stated that in the absence of splenic tissue, recurrences may be due to generalized reticuloendothelial cell hyperplasia and hyperphagocytosis in the liver, lymphatic system, and marrow. Neither theory can explain the recurrence of the capillary factor.

Since splenectomy is the recognized treatment for idiopathic thrombocytopenic purpura at present, certain preoperative criteria have been suggested in an attempt to prognosticate which patient will benefit by splenectomy. Doan[5, 7] advised the adrenalin biopsy test, but felt that a negative result in the presence of hyperplasia of qualitatively normal megakaryocytes is not a contraindication to splenectomy. Elliott[8] showed that his cases with low marrow megakaryocyte counts did not respond to splenectomy as well as those with normal or increased megakaryocyte levels. Thus normal or increased numbers of megakaryocytes indicate a good prognosis with splenectomy. Pernokis,[9] however, did not find increased megakaryocytes in his cases, and it may be inferred from his paper that effect of splenectomy in idiopathic thrombocytopenia cannot be foretold by means of sternal puncture. In the experience of most workers,[2, 3, 4, 6, 10] however, a good response to splenectomy is not obtained when marrow studies reveal a scarcity of megakaryoctes. Some have made statements to the effect that this finding should be considered a contraindication to splenectomy.

We wish to present a patient with idiopathic thrombocytopenic purpura in whom aspirated sternal marrow showed no megakaryocytes. Splenectomy was followed by a marked increase in peripheral platelets with disappearance of the bleeding tendency. A repeat marrow examination one year postoperatively revealed normal numbers of normal megakaryocytes. We believe this patient represents both a clinical and laboratory cure.

CASE REPORT

Mrs. L. M. (Register Number 81 472), a 26-year-old gravida zero housewife, was admitted to Madigan General Hospital Feb. 27, 1947, with a chief complaint of hemorrhages into the skin and mucous membranes of the mouth for several years.

She stated that since early childhood (4 years of age) she had always bruised easily and bled excessively with the slightest injury. She had noted frequent episodes of spontaneous epistaxis, and recurrent bouts of hemorrhages into the skin, mucous membranes of the mouth, and conjunctivae. Since the onset of menses she had experienced severe menorrhagia

which was unrelieved by various medications. In June, 1945, she underwent an oophorectomy for "cysts and tumors of the ovary." In October, 1945, a dilatation and curettage was performed to stop menorrhagia. This procedure was successful, but the next mensis was so severe that she underwent hysterectomy with removal of the remaining ovary as an emergency measure to check the vaginal bleeding. Since this last operation she took various estrogenic preparations irregularly for menopausal symptoms. This artificial menopause had no effect on the appearance of the purpura, and the bleeding tendency was not affected by any of the estrogens she took.

She had never noticed hemoptysis, hematemesis, hemarthrosis, melena, or hematuria. No history of chronic infection was elicited. She denied any exposure to arsenic, antimony, bismuth, iodine, benzol, di-nitro phenol, sulfa drugs, x-ray, radium, or other such substances capable of producing untoward effects on the hematopoietic system. The episode of purpura for which she entered began in October, 1946, and persisted until admission.

She stated that her father bled easily as a child, but this bleeding tendency disappeared in his later life. One sister has asthma. No other member of the family suffers with any other allergic condition. The patient herself did not complain of any manifestations of allergy or sensitivity.

Physical examination on admission was entirely normal except for extensive purpura and petechiae over the lower extremities, trunk, conjunctivae, and mucous membranes of the mouth. There was no icterus, lymphadenopathy, splenomegaly, or hepatomegaly.

The patient's clotting time was within normal limits (five minutes), but the bleeding time was prolonged to over ten minutes, and then stopped only by pressure. A Rumpel-Leede tourniquet test was strongly positive on multiple occasions. The clot retraction time was repeatedly prolonged, and on one occasion clot retraction was poor after seventy-two hours. Prothrombin times were normal. The hematocrit was 48; white counts and differentials were normal. All red cells and white cells appeared normal on smear. The fragility of the red cells was not increased. Repeated platelet counts fluctuated from 60,000 to 8,000 (normal by the method employed,[11] 200,000 to 500,000). Serology and urinalyses were negative. Examination of the stool and urine for blood was negative. Microscopic examination of aspirated sternal marrow on two occasions revealed no megakaryocytes after prolonged search, but the marrow was otherwise normal. Marrow smears were made from the first portion of marrow aspirated. On one occasion additional marrow was aspirated, centrifuged, and a smear made of the buffy coat. No megakaryocytes were found on any of these slides.

On April 8, 1947, under inhalation anesthesia, the patient underwent a splenectomy through a left subcostal incision. Moderate bleeding was encountered, and the patient was given a transfusion of 500 c.c. of blood during the operation. No accessory splenic tissue was found.

On the day of surgery, the platelet count was 30,000, and the bleeding time was five minutes. Platelet counts done twice daily on the first and second postoperative days ranged from 39,000 to 48,000. On the third postoperative day (April 11) the platelet count was 317,000 and the Rumpel-Leede tourniquet test was negative for the first time during hospitalization. The platelet counts continued to rise, and during the second postoperative week fluctuated from 900,000 to 1,260,000. During this time bleeding and clotting times were markedly decreased from preoperative levels. When the platelet count reached a million on the eighth postoperative day, the clotting time was forty-five seconds and the bleeding time was one and one-half minutes. During this period the patient was given anticoagulants; she developed no evidence of thrombosis. By the third postoperative week the platelet counts started to decline, and one month postoperatively, May 7, 1948, the platelet count was 375,000.

Follow-up.—Since the splenectomy, this patient was followed at monthly intervals for one year. She never noticed a recurrence of the previous bleeding tendency, and remained in excellent health. Repeated bleeding and clotting times, routine blood counts, and platelet counts stayed within normal limits. Examination of the sternal marrow in March, 1948

(one year postoperatively), revealed normal marrow with an abundance of normal-appearing megakaryocytes. This marrow was aspirated and prepared for microscopic study by the same technique as that used preoperatively.

Pathologic Examination of the Spleen.—The spleen was normal in size, shape, consistency, and appearance.

Microscopic examination was essentially normal and of no help in explaining the thrombocytopenia. In some areas of the pulp there were small accumulations of fat cells which were not xanthomatous. Numerous tissue eosinophiles were noted. A slight hyperplasia of the reticular elements was present.

DISCUSSION

No cause was discovered for the thrombocytopenia in this case. The finding of a normal spleen rules out the various causes of splenomegaly which can produce thrombocytopenia.[2, 5] The term "normal spleen" is used cautiously, because the spleen was not examined as thoroughly as in Doan's[5] discussion. This patient was not subjected to an adrenalin biopsy test. Comparative studies of the platelets in the splenic artery and vein were not performed. Excessive lecithinase activity of the spleen was not looked for. No supravital scrapings of the spleen were prepared to determine reticuloendothelial hyperplasia or hyperphagocytosis, although a "slight hyperplasia of the reticular elements" was found after reviewing a routine hematoxylin-eosin section.

No allergies or hypersensitivities were discovered in this patient. However, numerous tissue eosinophiles were observed in the spleen, and her family history revealed the presence of asthma. Schwartz[12] contended that a bone marrow eosinophilia in cases of idiopathic thrombocytopenia points to an allergic reaction of the megakaryocytes as the cause for the platelet reduction. These patients can be cured without splenectomy if the offending allergens are removed, but for unknown reasons they are cured by splenectomy also. The cases observed by Schwartz were usually of acute onset with spontaneous recovery. Our case does not fit into this category because of its chronic nature and the lack of both marrow eosinophilia and megakaryocytosis.

Watson[13] reported five cases of thrombocytopenic purpura following estrogen therapy, and suggested that some cases of idiopathic thrombocytopenic purpura occurring in menstruating women may be due to sensitivity to endogenous estrogen. In view of the fact that our patient since childhood had purpura which was unaffected by surgical removal of the ovaries, it does not appear feasible to believe that she was sensitive to any exogenous or endogenous estrogens. The skin was not tested with any estrogenic preparation.

Two preoperative sternal marrow studies in our patient revealed normal marrow except for the absence of megakaryocytes. No cause for this apparent megakaryocytic aplasia was found. There was no history of any dietary or vitamin deficiency, or of depressant chemicals, infections, or allergens. The marrow was not involved with any malignant infiltrative process, or by fibrosis. As mentioned previously, most workers would not expect the response to splenectomy which occurred; as a matter of fact, some would consider this finding in the marrow a contraindication to splenectomy. However, as stated by Richardson,[14] we wish to emphasize again the errors that may be derived

from any conclusions concerning a study of aspirated marrow. Megakaryocytes may be "absent" because of the dilution with blood that occurs during aspiration. Megakaryocytes may also be "absent" because they may have changed morphologically to resemble other stem cells. Furthermore, the marrow is a large organ, and its general condition may not be revealed by examining the area in the vicinity of a needle point. Both Richardson and Bauer[14] concluded that an actual marrow biopsy may be necessary to determine the true state of affairs.

The technique of making bone marrow preparations by utilizing isolated marrow units "fished" from citrated marrow is probably a better method to employ when searching for certain marrow elements such as megakaryocytes. This method would tend to obviate the errors that are possibly derived from studies of aspirated marrow diluted with peripheral blood. Unfortunately, neither this technique nor marrow sections were performed in this case. We feel, however, that the failure to find megakaryocytes after careful study of several slides (prepared from a centrifuged specimen on one occasion) may lend proof that in this case megakaryocytes were actually reduced. When one considers the various techniques that are employed in studying bone marrow, the difficulty in establishing what is the normal number of megakaryocytes is quickly apparent. Experience in such studies and careful examination of several smears made by various methods will be required, and will probably give more information than a mere differential count based on their frequency in relation to other cellular components.

Diggs and Hewlett[15] have published their observations that the number of megakaryocytes in smears of sternal punctures from patients with idiopathic thrombocytopenic purpura is not greater than that in a series of normal controls. Thus, the prognosis of splenectomy cannot be based on the number of megakaryocytes found in smear of marrow. However, these authors also observed that in idiopathic thrombocytopenic purpura the megakaryocytes were mostly immature without platelet production; megakaryocytes in normal controls were mature and showed platelet production. Thus prognosis of splenectomy is not to be correlated with the number of megakaryocytes, but rather on their appearance in thrombocytopenic purpura.

It is to be concluded that this patient's absence of marrow megakaryocytes was either a real absence of megakaryocytes, or that the techniques employed failed to demonstrate their presence. However, the fact remains that this patient with a scarcity of megakaryoctes (as determined on two occasions by commonly accepted techniques for marrow study) was cured by splenectomy, and examination of the marrow by the same technique one year later showed normal numbers of normal megakaryocytes. We feel from this experience that absence of megakaryocytes in aspirated marrow need not always be considered a contraindication to splenectomy.

SUMMARY

1. A brief discussion of idiopathic thrombocytopenic purpura is presented with review of opinions concerning the prognosis of splenectomy on the basis of marrow studies.

2. A case is reported in which studies of aspirated marrow prepared by commonly accepted techniques failed to reveal the presence of megakaryocytes, and in which a cure was obtained by splenectomy.

3. Difficulties encountered in studies of aspirated marrow are discussed.

4. Absence of megakaryocytes in aspirated marrow may not always signify contraindication to splenectomy when the history, physical examination, and other laboratory findings substantiate the diagnosis of idiopathic thrombocytopenic purpura.

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METASTATIC HYPERNEPHROMA OF THE THYROID GLAND WITH SUBSEQUENT THYROIDECTOMY, NEPHRECTOMY, AND RESECTION OF PULMONARY METASTASES

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THE thyroid gland is seldom the site of clinically evident metastatic cancer, and when a metastasis to the thyroid does produce signs and symptoms which focus attention upon the existence of a distant primary carcinoma, it presents an interesting clinical phenomenon. A recent experience with a renal-cell carcinoma, the presence of which was first manifested by the appearance of metastatic nodules in the thyroid gland, is the subject of this communication.

Less than twenty cases of metastatic hypernephroma of the thyroid gland have been reported in the literature. Prior to 1926 the metastases to the thyroid from hypernephroma were mainly incidental findings of post-mortem examination.[1-3]

In 1926, Kolodny[4] reported a case in which a clinical picture of hyperthyroidism (basal metabolic rate +80) caused the patient to have a thyroidectomy. Multiple nodules in both lobes of the specimen were described as metastatic hypernephroma. However, since then some doubt has arisen concerning this case since no primary tumor was demonstrated.

In 1934, Pemberton and Bennett[5] reported two cases in which the diagnosis of hypernephroma was made from tissue removed during thyroidectomy. In one case a man 71 years of age had a thyroidectomy for relief of a large nodular goiter which was causing choking and dyspnea. The patient died on the first postoperative day. Autopsy revealed hypernephroma in both kidneys and many metastases to pancreas, liver, and lymph nodes.

A second case was even more striking. This patient, a 38-year-old woman, was subjected to thyroidectomy because of the rapid increase in size of a goiter in two years' time. Pathologic examination of a 5 cm. tumor of the left lobe revealed metastatic hypernephroma in a colloid goiter. Following operation, an intravenous urogram was done, which showed an abnormality of the upper pole of the right kidney. A nephrectomy was performed and a small hypernephroma which had not penetrated the renal capsule was found. The operation was followed by a course of deep x-ray therapy. In a recent note, Pemberton[11] stated that this patient died May 15, 1943, or about ten years after the original operation, and, according to a letter from the local doctor who attended her shortly before her death, she was "suffering from a recurrent nodular swelling of the left lobe of the thyroid, vasomotor instability and has developed a psychosis—a retardation of both speech and action but only intermittently; however, becoming more severe."

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Since 1934, others[3, 6-8] have reported cases of metastatic hypernephroma to the thyroid gland. In most instances this occurred after an interval of several years following nephrectomy for the primary tumor.

H'Doubler,[3] in 1936, reported a case in which a metastatic hypernephroma of the thyroid was excised but, although the primary neoplasm was demonstrated by urograms, it was not removed and the patient died three years later from generalized metastases.

Boys,[9] in 1947, reported in detail a case in which metastatic hypernephroma was found in tissue excised during a thyroidectomy. The patient had had no symptoms related to the primary cortical carcinoma of the left adrenal which was demonstrated at post-mortem. Thus, this case was unusual in that the primary tumor was of adrenal cortical origin, apparently the only such case in the literature.

In the latest report[10] concerned with this subject, a thyroidectomy was performed on a patient who complained of swelling in the neck, dysphagia, and recent weight loss. Nine years previously a left nephrectomy was done for a renal-cell carcinoma. The patient had been well in the interim. Four and one-half months after thyroidectomy, at which time the diagnosis of metastatic renal-cell carcinoma was made, the patient was readmitted to the hospital complaining of pain and a mass in the right side of the abdomen. Surgical exploration of the right kidney revealed an inoperable renal tumor. Following a course of deep roentgen-ray treatment, he was discharged.

CASE REPORT

P. J., a 67-year-old farmer, underwent cholecystectomy for chronic cholecystitis and cholelithiasis on Jan. 12, 1947, at the University of Minnesota Hospitals. Following an uneventful recovery he returned to the outpatient department on May 5, 1947, for a routine postoperative examination. At this time a unilateral, firm, nodular mass was found in the right lobe of the thyroid. The examiner concluded that the nodule must have been enlarging rapidly, since previous examinations in January and February had not revealed its presence. The patient stated that he first became aware of a lump in the neck the previous evening. There were no signs of thyrotoxicosis. The basal metabolic rate was -18. Because of the apparent rapid increase in size, the patient was advised to have a thyroidectomy.

A total right thyroidectomy was performed* on May 21, 1947. The patient had an uneventful convalescence and was discharged.

The surgical specimen consisted of a thyroid lobe measuring 4 by 6 by 2.8 cm., containing approximately twelve rounded nodules of soft, gray-white tissue measuring from 1 mm. to 2 cm. in diameter. Microscopically, these were composed of polyhedral cells with clear cytoplasm and round hyperchromatic nuclei. The cells were arranged in cords, columns, and alveolar structures. The general appearance was strongly suggestive of a renal-cell carcinoma (Fig. 1).

A slide sent to the Army Institute of Pathology was reported upon as follows: "It certainly seems to be a metastatic hypernephroid carcinoma probably of renal origin . . . the possibility of a carotid body tumor should be ruled out."

The patient was readmitted to the hospital on July 21, 1947, for further study. Excretory pyelograms obtained at that time were considered "probably normal." Retrograde pyelograms (July 24, 1947) were also considered essentially normal.

*By Dr. Ivan D. Baronofsky.

Several observers noted a deformity of the middle minor calyx of the left kidney which was thought most likely due to a solitary cyst. The patient was discharged from the hospital with an appointment to return in three months for further consultation with the urological department.

After the three-month interval, urologic studies again demonstrated a persistent defect in the left kidney. The patient was admitted for exploration of the left kidney. At operation,* Nov. 3, 1947, two cysts were found occupying the lower pole, and perinephric fat was adherent to the kidney over the lateral border. When this was partly separated from the cortex a typical yellowish-appearing tumor was encountered; a nephrectomy was performed. No metastases were demonstrated.

Examination of the specimen revealed a rounded tumor mass measuring 5.5 by 5 by 3.5 cm. projecting from the mid-portion of the kidney laterally (Fig. 2). The cut surface was mottled grayish-white and moderately soft. It did not invade or deform the renal pelvis. Two thin-walled cysts measuring 4 and 1.5 cm. in diameter were present on the medial aspect of the lower pole and adjacent to the hilus respectively. Microscopic sections were characteristic of renal-cell carcinoma.

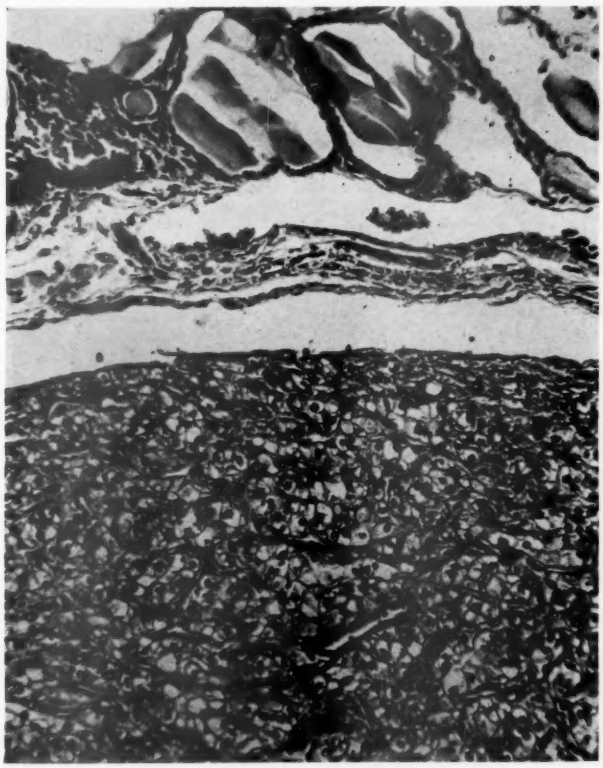

Fig. 1.—Portion of tumor nodule in thyroid gland. Thyroidectomy done May 21, 1947. Diagnosis was renal-cell carcinoma, metastatic.

The patient was discharged from the hospital on Nov. 12, 1947, and was seen at intervals in the outpatient clinic. His only complaint was of frequency of urination. When seen April 16, 1948, he had gained seven pounds in weight. However, a chest x-ray picture taken at that time revealed a rounded density about 2 cm. in diameter in the base of the left lung, and several areas of increased density in the right lung which were interpreted as possible metastases. Chest films were repeated during subsequent visits to the clinic (April 28 and May 18, 1948). On June 20, 1948, a fluoroscopic examination of the chest, as well as stereoscopic films, revealed again the 2 cm. nodular densities in the base of the

*Performed by Dr. George Eaves.

Fig. 2.—Kidney removed Nov. 3, 1947, showing the primary tumor, a typical renal-cell carcinoma.

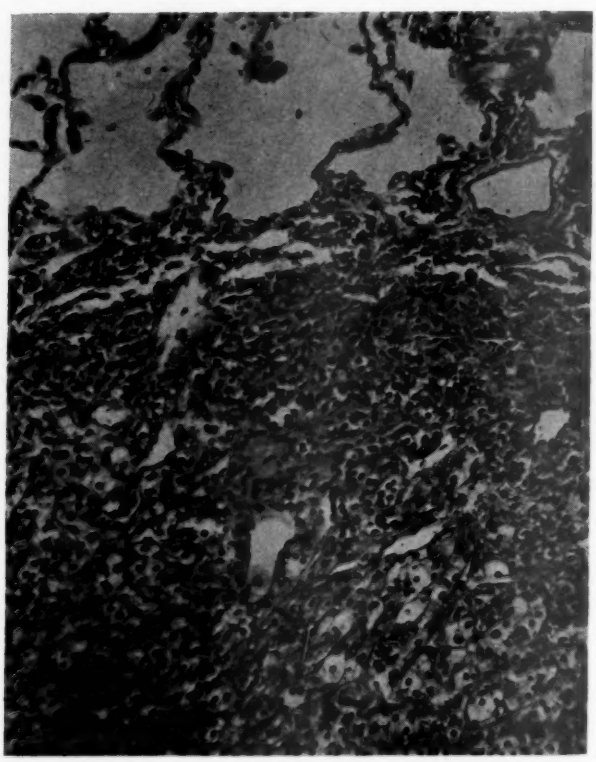

Fig. 3.—Microscopic section of pulmonary tissue and one of the three nodules removed by segmental resection on June 20, 1948. Diagnosis was renal-cell carcinoma, metastatic.

left lung. The right lung appeared clear. In view of the fact that no other metastases had developed and those present in the lung had not enlarged markedly in three months, the patient was readmitted for an exploratory thoracotomy and possible segmental resection of the metastatic lesion.

On June 20, 1948, one year after the thyroidectomy which disclosed metastatic hypernephroma, and six months following removal of the primary tumor, the patient was again subjected to surgery.

The operative procedure* was as follows. An incision was made through the seventh interspace on the left. A 3 cm. nodule could be palpated in the periphery of the basal segment of the lower lobe. Close, but distinctly separate, were two other smaller nodules. All three nodules were removed by small segmental resection between angled hemostats. A fourth nodule on the lung surface was also removed although it did not have the appearance of tumor tissue. A catheter was placed to make certain that no tension in the thorax developed. The chest wall was closed with interrupted silk.

Examination of the fragments of pulmonary tissue revealed three tumor nodules measuring from 2 mm. to 2 cm. in diameter. The fourth nodule was calcified. Microscopically, the structure of the three tumors was that of a renal-cell carcinoma (Fig. 3). Eight months after thoracotomy the patient was free of symptoms and repeated chest x-ray examinations have not disclosed further metastases.

DISCUSSION

This case is the seventeenth case to be reported in the literature of a renal-cell carcinoma which had metastasized to the thyroid gland. This is in addition to the case of a metastatic adrenal carcinoma reported by Boys. In only one previous case[5] was the primary lesion removed after thyroidectomy disclosed metastatic hypernephroma. The extended period of palliation that was obtained in Pemberton's case (ten years) certainly would seem to justify removal of the primary tumor.

The preferential growth of metastases in various organs has been discussed many times. The occurrence of multiple metastases in the thyroid would indicate that probably many organs received tumor emboli but that only under the most favorable conditions did they grow. In our case, a group of pulmonary metastases suddenly enlarged enough to be detected by roentgen examination one year after the metastases to the thyroid had been resected, and four months after the primary lesion had been removed. Apropos are the cases cited previously in which thyroid metastases were present many years before or after similar phenomena affected other organs. The second case reported by Pemberton is an unusual example of this process.

Thus, the disclosure of metastatic hypernephroma of the thyroid with no other obvious metastases should not deter the surgeon from attempted removal of the primary tumor. The natural history of hypernephroma is such that prolonged palliation, survival, and perhaps even cures may result from aggressive surgical therapy of this tumor.

SUMMARY

A case of hypernephroma (renal-cell carcinoma) first manifested by thyroid metastases is presented. Subtotal thyroidectomy, nephrectomy, and segmental resections of pulmonary metastases were performed.

*Operation performed by Dr. R. L. Varco.

ADDENDUM

Subsequent to the submission of this paper for publication, the patient developed symptoms of an intracranial lesion. Studies revealed a lesion in the left retrobulbar area. Neurosurgical consultants did not deem surgery advisable; therefore, he was treated by deep x-ray therapy (1,200 r/a to three fields). The patient expired in December, 1949.

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PHEOCHROMOCYTOMA, WITH PERSISTENT HYPERTENSION, AND THORACOLUMBAR SYMPATHECTOMY

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IN THE earlier concepts of the clinical syndrome associated with pheochromocytoma, paroxysmal hypertension was thought to be the principal manifestation. In the last few years, however, many reports have been made describing pheochromocytoma accompanied by persistent hypertension and even simulating malignant hypertension.[1-4] Therefore, the likelihood of some confusion of this syndrome with that of essential hypertension exists, and care in the differential diagnosis is indicated. It has been suggested that pheochromocytoma at least be considered in each case of severe hypertension[5]; in the light of the following case, we believe this to be true, especially before surgery for hypertension is attempted. This incidence, however, of functioning pheochromocytoma in hypertensives is small. Smithwick[6] has reported an incidence of 0.4 per cent in hypertensive patients who came to surgery.

We now have better means of diagnosing adrenal medullary tumors, such as the use of intravenous or retrograde pyelograms, x-ray films after perirenal insufflation of air, the histamine test,[7] and the use of an adrenolytic benzodioxane such as 1164 F in a test for circulating epinephrin.[4-8] These, together with the history and physical examination, should make the diagnosis less difficult.

It is the purpose of this paper to report a case in which pheochromocytoma remained undiagnosed, the patient being treated for arterial hypertension by sympathectomy. So far as we have been able to ascertain, the only other reference to such a case was made by Binger and Craig,[10] who reported a case where a pheochromocytoma was removed ten days after sympathectomy.

CASE REPORT

Case Summary From Another Hospital.—F. C., a white man 25 years of age, dated his illness from 1944, when he was in the Army. At this time, following an automobile accident, he began to have severe headaches and episodes of vomiting. He was hospitalized and was found to have hypertension. Because the symptoms persisted, he was eventually discharged from the Army with a diagnosis of essential hypertension.

On Dec. 3, 1947, he was hospitalized again with the same complaints. The headaches were varying in degree, often very severe, and sometimes lasted several hours or even days. He complained of bouts of rapid heart action, and he was distinctly heart conscious. He stated that while at home he had a voracious appetite. Past history revealed no injuries or operation, but twelve years before he had had a swollen testicle. The family history was negative.

Physical examination at this time showed a young man only fairly well nourished and developed. The heart and lungs seemed normal. The blood pressure ranged from 160 to 240 mm. Hg systolic and 100 to 190 diastolic. The pulse rate was seldom below 90 when taken repeatedly over several days. There were marked vascular changes in the optic fundi. X-ray examination showed the heart and lungs to be normal. Blood counts and chemistry, serology, and urinalyses were normal. The electrocardiogram was interpreted as showing possible myocardial damage (deep S_3 and inverted T_3). A retrograde pyelogram and renal

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function tests were considered normal. Response to a sedation test was poor; the blood pressure dropped from 180/135 to 160/118 and quickly rose again. The basal metabolic rate on two occasions was + 22 and + 5.

The patient was seen by a consultant in neurosurgery and judged to be a fairly good candidate for sympathectomy. On Jan. 20, 1948, a left sympathectomy from the seventh thoracic vertebra to second lumbar vertebra was done. The adrenal gland was palpated during the operation and was thought to be normal. The blood pressure did not change appreciably. Accordingly, on Feb. 3, 1948, a similar procedure was done on the right. Immediately following both operations, some hypotension developed which required Adrenalin medication. That following the second operation was most severe. In general the blood pressure values were now much lower than formerly (average 140/110), although isolated instances of high blood pressure were recorded. This response was considered "gratifying."

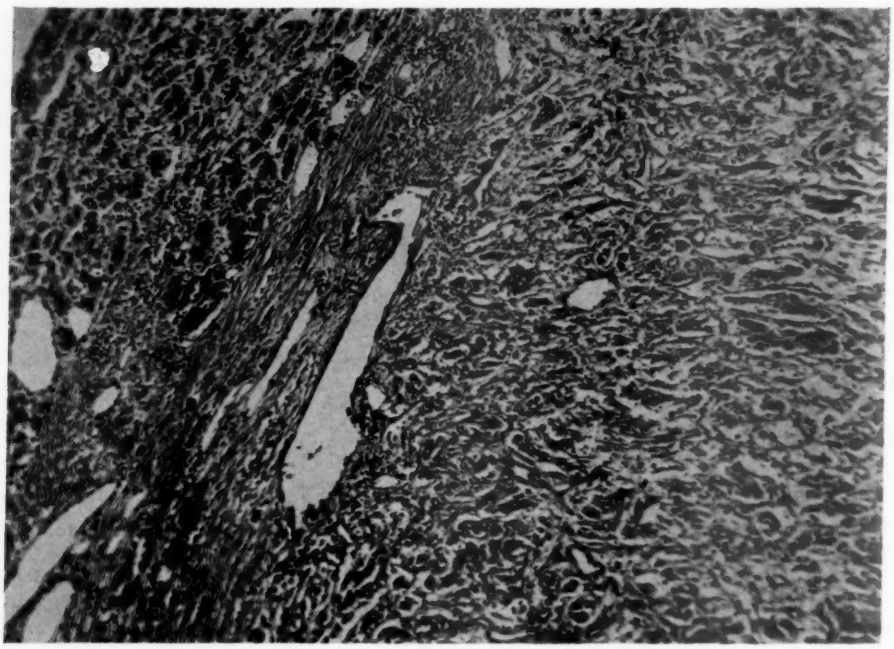

Fig. 1.—Normal adrenal cortex is seen in the upper left, and tumor tissue (pheochromocytoma) is seen on the right. A fibrous capsule is present.

After surgery, however, the patient cooperated poorly, ate poorly, and complained continually of pain in the chest and about the operative site. He was reluctant to sit or stand because of dizziness. There developed profuse sweating of the trunk, face, neck, and arms, and marked tachycardia. He was given elastic supports for his legs and abdomen, but he refused to wear them. Large amounts of sedation were given because he complained continually. After a few days' convalescence he was discharged from the hospital, but at home he was no better. The care of his family was required constantly.

Course at Franklin Square Hospital.—After his discharge from the hospital mentioned previously, the patient remained in an invalid state. Two days before admission to this hospital (May 2, 1948) he began to develop the additional symptoms of cough and dyspnea. These symptoms became progressively worse, and a few hours before admission he passed into cardiovascular collapse. On arrival at the hospital he appeared moribund. The skin was cold and clammy. The respirations were rapid. The temperature was 101.0° F., rising to 103.6° in a few hours. Coarse râles were heard throughout both lungs. He had no measurable blood pressure. The heart was enlarged. The remainder of the physical examination was negative.

Because exodus seemed imminent, treatment was instituted immediately. He was given oxygen by mask, strophanthin, Salyrgan-theopylline, Adrenalin, cortin, and plasma. Recovery from shock was dramatic and surprisingly rapid. Four hours later, he had recovered enough to answer questions, and seemed no longer to be in any danger. The blood pressure steadily rose, first to normal, and in the ensuing days to a maximum of 190/140. The pulse rate varied widely daily from 80 to 130, but usually was over 100. The signs and symptoms of profuse sweating, tachycardia, pallor, constipation, abdominal pain, and prostration which he had soon after admission continued until his death. On the day before death, the patient became quite dyspneic and complained of severe pounding of the heart. A split first sound was heard at the cardiac apex at this time, and the blood pressure was 190/140. On the next day, he had a convulsion, followed rapidly by death. Intracardiac Adrenalin was of no avail. A blood pressure reading during this attack was not obtained.

Autopsy.—The main pathologic findings at autopsy were: cardiac hypertrophy; arteriosclerotic changes in the coronary arteries, the aorta, and in the aortic and mitral valves; and a tumor of the left adrenal gland. This tumor was soft, dark, and 4 cm. in diameter. The cut surface was yellowish. A distinct green color was obtained when a solution of ferric chloride was applied. Microscopically nothing unusual was noted except in the adrenal. Sections of the tumor showed cells of a new growth arranged in clusters, a fibrous capsule was present, and normal adrenal cortex was seen outside the capsule (Fig. 1). Sections stained with Schmorl's reagent showed the dye taken up as greenish-brown granules by the cytoplasm of the neoplastic cells.

DISCUSSION

Sympathectomy in the presence of a functioning pheochromocytoma is an unusual pathologic situation, although Smithwick believes it is more common than reported.[11] In this patient, although a seemingly good result as to blood pressure readings was obtained at first, the combination proved incapacitating. Following the operation he no longer had persistent hypertension, but he was subject to the extremes of blood pressure variance. He had the postural hypotension which follows sympathectomy and also the attacks of hypertension due to excessive adrenalin secretion. He was prostrated, and from then on his general condition deteriorated.

The symptoms for which the patient was admitted to this hospital, however, seemed unrelated to sympathectomy. He had, evidently, developed an upper respiratory infection and a superimposed epinephrine shock. He was recovering well from this episode only to have another cardiovascular crisis which was fatal. Both of these attacks seemed due, primarily, to pheochromocytoma and probably would have occurred regardless of sympathectomy.

SUMMARY

1. The differential diagnosis of persistent hypertension or paroxysmal hypertension should include pheochromocytoma.

2. A case of pheochromocytoma, in which a thoracolumbar sympathectomy had been performed, is presented with the clinical and pathologic findings.

3. Careful palpation of the adrenal glands should be carried out during the procedure of thoracolumbar sympathectomy in order that this adjuvant to the treatment of arterial hypertension may not be misused.

Addendum.—For a somewhat similar report, see Hatch and associates.[12]

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A RARE CASE OF BACILLUS PYOCYANEUS (PSEUDOMONAS AERUGINOSA) PYOARTHROSIS OF THE KNEE

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THE genus pseudomonas is a group of gram-negative bacilli that produce water-soluble pigments. *Pseudomonas aeruginosa* was named *Bacillus pyocyaneus* by Gessard[1] in 1882, and it is the only pseudomonas pathogenic for man. Pyoarthrosis caused by this organism is a rarity.

HISTORICAL REVIEW

A review of the literature reveals few other cases of joints affected by this organism. Bishop[3] described the case of a 3½-year-old child who had sustained burns on the body, including the right leg and foot, one and one-half years prior to admission to the hospital. Culture from the burned area revealed *B. pyocyaneus* infection. An abscess over the insertion of the tendon Achilles which developed later also showed a pure growth of *B. pyocyaneus*. X-ray examinations indicated destruction of the lower right tibial epiphysis. The child was treated with an autogenous vaccine and had an uneventful recovery. Groves[2] reported a secondary infection of a hip joint from which *B. pyocyaneus* was isolated following an operation for tuberculosis in an 8-year-old boy. A septic course of two months' duration ensued, and purulent arthristis of the right thumb and the opposite hip developed. *B. pyocyaneus* was isolated only from the original site. The other sites were not cultured, but it is likely that the other joints involved were metastatic. Autogenous vaccine was given and the boy recovered. The case of an 8-month-old baby who developed acute symptoms of the left shoulder from which *B. pyocyaneus* was aspirated was described by Pinelli.[4] A vaccine made from the aspirated material was given and the patient recovered. Melina[5] reported upon a 10-year-old girl with a pleural effusion who later developed destruction of the acetabulum in the head of the right femur. Aspiration of this hip yielded a purulent material from which *B. pyocyaneus* was isolated. An autogenous vaccine was made and administered, and the patient recovered. Perkins[6] reported a 30-year-old man who had developed an abscess near the left seventh rib, and an acute purulent arthritis of the left elbow following a pneumonia with pleural effusion in the left pleural cavity. *B. pyocyaneus* was isolated from both areas. This patient died. Waite[7] described the case of a 20-year-old woman who developed acute symptoms of the left knee. The origin of the infection was not known. Treatment was amputation of the extremity. A case was reported by Fiset[8] of a 77-year-old man who had a chronic multiple arthritis caused by this organism which was isolated only from the stools. The patient was successfully treated with an autogenous vaccine.

CASE REPORT

F. W. H., a 58-year-old white man, an electrician, was hospitalized on Jan. 11, 1947, for an adenocarcinoma of the rectum. He complained that he had had slight rectal bleeding for

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one year and a loss of about ten pounds in weight. On Feb. 1, 1947, an abdominal perineal resection of the rectum was performed. He was given a number of transfusions postoperatively. A yellowish drainage from the perineal wound, noted on the fourth postoperative day, persisted despite daily irrigation. *B. pyocyaneus* was cultured from the wound. Fever from 100 to 101° F. was present daily. On Feb. 19, 1947, the temperature spiked to 102° and continued to range between 101 and 103° F. On Feb. 25, 1947, the patient was seen for the first time by the orthopedic department because of persistent temperature of over 102°, and pain in the left knee. The knee was markedly swollen, tender, and painful on

Fig. 1. Fig. 2.

Fig. 1.—Anteroposterior view of left knee, taken on March 21, 1949.
Fig. 2.—Lateral view of left knee taken on March 21, 1949, showing osteophytes of patella.

motion. A thick, yellowish, purulent material was obtained by aspiration, and the specimen was sent to the laboratory for culture. The knee was irrigated daily with penicillin solution until March 3, 1947, when the solution returned clear. From 1,000 to 1,500 c.c. of solution containing 200,000 units of penicillin per 500 c.c. of solution were used. Penicillin was employed because the nature of the organism was not known at the time. Traction with a five-pound weight was applied to the extremity. Four cultures, taken on Feb. 25, 26, and 27, 1947, and March 15, 1947, were reported as pure growths of *Ps. aeruginosa*. The first culture was recorded from the laboratory on March 3, 1947. For this reason, from that date on, streptomycin was used for daily irrigation instead of penicillin, using 1 gm. of streptomycin per 1,000 c.c. of solution. Also, 1 gm. of streptomycin daily was given parenterally. The day following the first irrigation of streptomycin the drainage was thinner and less profuse. Throughout the course of illness, there was a thick, fibrinous, sticky material that could not be removed completely by irrigation. From March 10, 1947, to April 8, 1947,

irrigations were carried out every other day. The patient received a total of twenty-six irrigations, seven with penicillin and nineteen with streptomycin. During the course of treatment, he was instructed to contract the quadriceps hourly. From April 8, 1947, on, he was under the guidance of the physiotherapy department, and was started on active exercises for the knee. These exercises were gradually intensified. At the time of discharge from the hospital on Sept. 10, 1947, painless motion was present from 70 to 180 degrees. No instability of the knee existed. He had discarded crutches, and a limp was barely perceptible. This patient was last seen in April, 1949. He had gained weight and had no complaints referable to the knee or to the residuals of surgery. Knee motion was present from 50 to 180 degrees and the limp had disappeared. No atrophy of the quadriceps existed. X-ray pictures revealed small osteophytes on the posterior margin of the patella but no other changes (Figs. 1 and 2). Because of the return of good painless motion, it is not expected that the patient will have any future disturbance at this joint.

TABLE I. LABORATORY FINDINGS

DATE	LOCATION	SMEAR	CULTURE
2/15/47	Perineal wound	No organisms or fungi	*Ps. aeruginosa* colonies
2/25/47	Knee	No organisms or fungi	*Ps. aeruginosa* colonies
2/26/47	Knee	No organisms or fungi	*Ps. aeruginosa* colonies
2/27/47	Knee	No organisms or fungi	*Ps. aeruginosa* colonies
3/15/47	Knee	No organisms or fungi	*Ps. aeruginosa* colonies

2/17/47 R.B.C. 3,930,000; hemoglobin 12 Gm. per 100 c.c.
Urinalysis: albumin, positive; microscopic, few pus cells; specific gravity 1.020; glucose, negative
Kahn: Negative

CONCLUSION

A rare case of *B. pyocyaneus* pyoarthrosis of the knee is presented. It is only the third reported case of a pyoarthrosis caused by this organism in an adult and the first case reported that has been successfully treated with streptomycin. The excellent result obtained is attributed to the administration of streptomycin parenterally, and to frequent irrigations of the joint with large amounts of saline solution into which streptomycin had been instilled.

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Editorial

A New Approach to the Treatment of Burns and Scalds

IN THE United States there are about 5,000 deaths a year from burning accidents (excluding conflagrations, which are responsible for about 2,000 more).

The number of nonfatal, though serious, accidents is not known, but on a conservative estimate it may be put at approximately 100,000 (5,000 × 20). Taking that figure as the probable number of patients admitted to hospital wards, and the average length of stay at fifty days, the usage of hospital facilities on these injuries must amount to something like five million bed-days.

These large figures compel us to ask—Could the number of these deaths be considerably reduced? And could the care of patients with nonfatal injuries be improved so that their slow healing is speeded up, and the unhappy results of slow healing are largely eliminated?

An American surgeon discussing these questions recently stated categorically that burns have been more mismanaged than any other kind of injury; I think most conscientious surgeons in every country would agree with him. We must admit that few burns have had the best treatment that medical science could provide. Infection of the extensive burn has been regarded as inevitable, and until recently no one has seriously attempted to apply to these injuries the Listerian principles of asepsis which were so brilliantly successful in controlling the septic infections of the operating theater. The treatment of "burn shock" has also left very much to be desired in most hospitals; often it has been carried out by quite inexperienced officers.

How can all this be remedied and a new standard created? One remedy is suggested in a book recently published under the title which stands at the head of this editorial.* It is proposed that we should abandon the time-honored plan of admitting burned patients into every general hospital—and into "skin hospitals" as is often done on the continent of Europe—and should create instead one or more special centers in all large cities, where special equipment and a trained staff would always be available for dealing with them under optimal conditions. Such centers should work in the closest cooperation with an applied bacteriologist having adequate laboratory facilities at his command.

Experience of such a special center, established in 1944 by the Medical Research Council at the Accident Hospital of Birmingham, England, has been freely drawn upon in putting forward these suggestions—in particular the remarkably happy results obtained by the plan of dressing all burns in an atmosphere almost microbe-free.

*Colebrook, Leonard: A New Approach to the Treatment of Burns and Scalds, London, 1950, Fine Technical Publications.

This atmosphere has been provided by bringing into the special dressing room an abundant stream of twice-filtered and warmed air, which mechanically flushes away any particles carrying bacteria such as are liberated during each dressing. By new methods of sampling the bacterial content of the air from minute to minute during the course of a dressing it has been shown conclusively that this scatter of microbes is often very large, particularly while bandages are being removed, and that the microbes so scattered frequently include large numbers of "wound pathogens."

The Birmingham experiment is only one step toward the ideal treatment of the burned patient. The buildings available there do not provide the best possible conditions but the experiment has demonstrated beyond any doubt that the outlook for such patients is entirely changed when sepsis is prevented and controlled. Pain, and the terror of dressings, can be almost eliminated from 90 per cent of the patients, and with them the distressing "personality changes" so often seen in the past; the period of incapacity can be greatly reduced (often by 50 per cent or more) if early grafting of the deeply burned area is practiced, as it can be, without fear of septic complications; the contractures and consequent economic loss that have clouded the lives of so many burned children need be seen no more.

These great gains will not be won easily. In the nature of things, it must always be difficult to avoid infection of the extensive burn. But it can be done. It will not be done unless we take trouble—and in the right way.

—*Leonard Colebrook.*

Should Patients Be Told They Have Cancer?

THE most important person in an illness is the patient himself. Sometimes this is forgotten by the physician as well as relatives of the patient. Straightforward honesty and sincerity are traits which we appreciate in transactions with our fellow men and take for granted in our dealings with intimates. The physician-patient relationship is one which presumes trust, sincerity, faith, and confidence. And yet, how often we violate this trust when informing patients who have cancer concerning the nature of their illness. But why?

When Babe Ruth,* colorful hero of baseball, died, a well-known radio commentator said the nature of Ruth's illness had been so carefully hidden from him that he probably never knew he had cancer. One would have to be extremely gullible to believe that.

Yet, physicians frequently follow a policy of dissimulation in acquainting patients with the nature of a cancerous malady. We do not deliberately deceive the patient or are not guilty of intentional fraud, but the deception is nevertheless a violation of that sincerity and trust which the patient rightfully may expect of his physician.

A critical examination of what we are accomplishing by such artifice is very much in order. I am guilty of having employed this strategy in earlier years in an attempt to evade informing patients who have cancer concerning the true nature of their illness. Nevertheless, I am persuaded that, though the word cancer may never have been spoken within the patient's hearing, yet the patient usually divines what we are striving to hide from him, particularly if the treatment carried out is only palliative. In fact, he too often becomes a party to the conspiracy, being careful not to let relatives or his physician learn that he (the patient) knows he has cancer.

What is the effect of such deception? In the first instance, it makes the patient mistrust his physician; moreover, it becomes difficult for him to understand the insincere behavior of his family. Many a patient, who has been caught up in this plot of deceit and has become fully aware of the circumstance that his strength is failing and nothing can be or is being done to help him, probably says to himself, "Why do not my physician and my relatives tell me the truth? Why do they lie and attempt to deceive me? They know very well what is the matter with me; they are blind if they do not perceive that I know as well as appreciate that they know. Why this atmosphere of faithless fraud? By their attempts at deception, my friends have destroyed what might have been a beautiful relationship in my final illness." In his loneliness, bitterness, resentment, and self-pity are natural outlets of relief for the unhappy patient.

Why do physicians and relatives conspire to try to hide from patients that they have cancer? Whatever the contributory causes may be, some of which will be mentioned presently, the real reason is undoubtedly an attempt to protect the patient from anxiety, fear, and worry, and to protect the family

*Babe Ruth (George Herman Ruth) died Aug. 16, 1948.

from needless uneasiness. We have already seen what this organized deception may do to the patient. Moreover, the endeavor to conceal from the patient the true nature of his illness is done probably as much with a view to protection of the family as the patient. Life must go on, and anything done with a view to continuing a resemblance of a normal mode of life is justified as being in the best interests of the patient. In other words, to tell the patient that he has cancer may upset the family routine, making it mandatory for the family and the patient to effect an adjustment in their relationship before it would otherwise have been necessary.

It is perhaps not out of place to suggest that meeting the convenience of the family in this matter is, in the end analysis, probably the item largely responsible for justifying the conspiracy of not telling the patient he has cancer. Leo Tolstoy depicts this situation well in his story of "The Death of Ivan Ilich" written in 1866. Everyone, including the patient, knew he was dying; but his family, mindful of their own interests, affected to have Ivan believe that his illness was not serious. Counterfeit behavior is not difficult to detect; even a sick man distinguishes easily in his associates the assumption of a light and Pollyannish air toward a matter which he knows intuitively is far more grave. Moreover, the very veering about in attitude of the relatives, from one of urging a concern on the part of the patient in his own illness, to one of affected unconcern on their part, affirms in his mind the certainty of the seriousness of his disorder, whatever it may be.

It is not easy for anyone to share wholly the trials of another. History records no better example than that of Jesus with His disciples, Peter, James, and John, at Gethsemane. While the Master prayed for deliverance, they slept. Little wonder He was provoked to say: "The spirit indeed is willing, but the flesh is weak." These words have immortalized Gethsemane and made it a byword in our language. Yes, it is difficult to share fully another's Gethsemane, as everyone knows.

The preceding lines were written two years ago, directly after hearing the radio commentator speak of Babe Ruth's illness. They relate essentially to the far-advanced case. If one can justify to himself, let alone to others, telling a patient with late cancer, for whom one can do little, that he (the patient) has cancer, then what of the early case? Recently we set about in this clinic to ascertain what patients thought about all this. Elsewhere in this JOURNAL there is a brief report of an interesting study by Doctors William D. Kelly and Stanley R. Friesen. It is important to find out what patients think as well as what we do about this important matter. The answer is very plain; patients in the Cancer Detection Center Group, those who come to the outpatient clinic of the hospital, as well as those who come because their home physicians believe or suspect the patient has cancer—the majority of all these patients want to be dealt with sincerely and honestly. In other words, if, during the examination, cancer is discovered they expect and want to be told the truth.

Yet highly reputable surgeons fail to tell their patients they have cancer. And from discussions with physicians and roentgenologists, as well as surgeons

in several areas of this country, I infer that an assumption of an evasive attitude is common practice. In *McCall's Magazine* for February, 1950, there was an article on this discussion concerning breast cancer. It was a dialogue between a well-known American surgeon, Dr. Frank Adair of the Memorial Hospital in New York, and Mr. John Gunther, a modern writer of distinction, whose sensitive portrayal of his own son's illness in *Death Be Not Proud* touched the hearts of everyone who read it. Dr. Adair defended the thesis of not telling patients they have cancer and said: "I never use the word 'cancer' at all."

Now this consideration is of far greater import than mere differences in philosophic outlook. There is need, I believe, for a bolder, a more sincere and realistic attitude—yes, a more enlightened view toward cancer in dealing with our patients.

In what other disease do we fail to acquaint patients with the nature of their illness? None that I know of. Is the discovery of cancer still synonymous with a sentence of doom? Certainly not, unless the cancer has been neglected. I have heard a prominent university professor in public health say "Cancer may turn out to be a way of life." I do not feel that pessimistic over the outlook in cancer, nor do I believe there is any justification for such a belief.

Cancer is curable. Whereas our main problem is early recognition, treatment of the late cancer will probably always be with us. While we strive to focus important notice upon early detection of the presence of cancer, we must continue to improve our management of the late case. Important progress in the treatment of cancer has been made during the past few years, essentially through combined and concerted efforts in stressing to the public the importance of early diagnosis. In gastric, colic, or rectal cancer, a patient with symptoms already has a cancer which is approximately twenty months old. If four months or more (an interval which is not unusual) intervenes before the patient seeks treatment, the cancer has been developing for about two years by the time the patient reports for treatment. Hence it is of great importance to develop screening tests for cancer, and to have periodic careful examination of patients above 45 years of age, when cancer begins to take a larger toll in both sexes. Will the time ever come when we will recognize the majority of the frequent cancers of the colon and rectum, stomach, breast, and uterus when these lesions are still local, without lymph node metastases? I do not know. I would like to think so. Certainly, if we continue to await the occurrence of symptoms, we shall continue dealing with late cancers—patients whose regional lymph nodes are involved, and in whom the cure rate, as contrasted with patients without lymph node involvement, drops from 75 to 20 per cent for the breast, colon, and rectum.

In the state of Connecticut, the five-year cure rate for all cancer is reported as being 33.5 per cent.[4] This is a record of substantial accomplishment. It sets a high standard for states with a less crystallized program and interest in cancer. And yet that record too, I believe, will be improved upon presently.

The majority of patients with cancers upon whom we operate have lymph node metastases (approximately 75 per cent). With reference to colon, rectal, stomach, and breast cancers with lymph node involvement in our clinic, my colleagues and I have come to the point of view that such patients should undergo reoperation for a "second look" within four to six months.[5] In the visceral cancers treated in this manner, 75 per cent have been found to have cancer at the time of the "second look." In a few patients, we have done a third "second look"; to one patient a fourth "second look" has been proposed and will be carried out, because in the third "second look," two small cancerous periaortic nodes were found, the initial operation being done for a huge cecal carcinoma with widespread mesenteric lymph node involvement. Our objective in such patients is one "second look" in which no cancer is found.

Obviously, it would be absurd to fail to tell the patient he has cancer. If we want the patient's cooperation, which we must have, of necessity, he must be told. A well-known judge in my community came to talk with me about his operation a few weeks after leaving the hospital. He said, "Did I have cancer?" My answer was in the affirmative. I then countered by saying, "When did you first learn?" He said, "I looked at the chart."

We are all being examined by judges in this matter every day, perhaps not by judges of the law but by persons whose clarity and acuity of vision in this important matter transcends our own. To fail to tell patients they have cancer is as archaic and outmoded as Victorianism.

—*Owen H. Wangensteen.*

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Book Reviews

Practical Neurological Diagnosis. By R. Glen Spurling, M.D. Ed. 4. Pp. 258, with 101 illustrations. Springfield, Ill., 1950, Charles C Thomas, Publisher. $5.

This is a very simple outline of the general procedures followed in a routine examination of the nervous system, including some explanation of the principles behind these procedures. The simplicity of this text certainly limits its use to the beginning student. The author has perhaps oversimplified many features in the neurological examination. This is particularly true in his discussion of history-taking, where such important symptoms as paralysis, weakness, dizziness, visual disturbances, and mental disturbances are completely omitted. A good share of the emphasis is placed upon those symptoms observed in the expanding lesions of the intracranial cavity, which actually comprise but a small part of a neurological diagnosis. Similar omissions are found in the discussion of the symptomatology of corpus striatum lesions where such important disturbances as chorea and athetosis are not even mentioned.

In the neurological examination the nerve supplies of the various muscles are listed but no attempt is made to describe the methods used in examining and evaluating disturbances in the strength of the various muscle groups.

This small volume should prove quite useful as a preliminary survey of the neurological examination in spite of its incompleteness. Its oversimplification, however, may leave an incorrect concept of the many detailed procedures that are necessary to detect adequately disturbances in nervous system function.

Fractures and Orthopaedic Surgery for Nurses and Masseuses. By Arthur Naylor. Ed. 2. Pp. 296, with illustrations. Baltimore, Md., 1948, Williams & Wilkins Company. $5.

This small volume attempts to compress and simplify a tremendous amount of material on traumatic and orthopedic subjects. The author has accomplished his purpose remarkably well. Specific deficiencies exist, however, such as the recommendation of local implantation of sulfonamide powder in wounds, and the discussion of the use of antibiotics generally. The value of this book lies chiefly in its fine illustrations and diagrams, which have been carefully culled from major works on traumatic and orthopedic surgery. These can be easily integrated in any teaching program for nurses and technicians, making this volume especially valuable for nursing supervisors and teachers.

Books Received

The receipt of books is acknowledged in this section and this treatment must be regarded as sufficient acknowledgment of the courtesy of the senders. Selections will be made for more extensive review dictated by the interests of our readers and as space permits.

ESSENTIALS OF ORTHOPEDICS. By Philip Wiles, M.S. (London) F.R.C.S. (Eng.) F.A.C.S., Hon. Orthopedic Surgeon, Middlesex Hospital and King Edward Memorial Hospital. Cloth. Price $10. Pp. 486, with 372 illustrations. Philadelphia, 1949, The Blakiston Company.

TEXTBOOK OF NEUROPATHOLOGY. By Ben W. Lichtenstein, B.S, M.S., M.D., Associate Professor of Neurology, University of Illinois College of Medicine. Cloth. Price $9.50. Pp. 474, with 282 illustrations. Philadelphia, 1949, W. B. Saunders Company.

REGIONAL ILEITIS. By Burrill B. Crohn, M.D., Consulting Gastroenterologist, Mount Sinai Hospital, New York. Cloth. Price $5.40. Pp. 229, with 74 illustrations. New York, 1949, Grune & Stratton, Inc.

FUNDAMENTAL CONSIDERATIONS IN ANESTHESIA. By Charles L. Burstein, Chief, Department of Anesthesiology, Hospital for Special Surgery, New York. Cloth. Price $4. Pp. 153, with 64 illustrations. New York, 1949, The Macmillan Company.

BLOOD TRANSFUSION. By Geoffrey Keynes, M.A., M.D., F.R.C.S., St. Bartholomews Hospital, London, and others. Cloth. Price $12.50. Pp. 574, with 109 illustrations. Baltimore, 1949, Williams & Wilkins Company.

THE DIAGNOSIS OF PANCREATIC DISEASE. By Louis Baumann, M.D., Presbyterian Hospital, New York. Cloth. Price $5. Pp. 74. Philadelphia, 1949, J. B. Lippincott Company.

OPERATIONS OF GENERAL SURGERY. By Thomas G. Orr, M.D., Professor of Surgery, University of Kansas, Kansas City, Kan. Cloth. Ed. 2. Pp. 890, with 1700 illustrations. Philadelphia, 1949, W. B. Saunders Company.

ORTHOPEDIC SURGERY. By T. P. McMurray, C.B.E., M.B., M.C.H., F.R.C.S. (Edin.) Professor of Orthopedic Surgery, Liverpool University England. Cloth. Price $8. Ed. 3. Pp. 444, with 191 illustrations. Baltimore, 1949, Williams & Wilkins Company.

ILLUSTRATIONS OF SURGICAL TREATMENT. By Eric L. FarquLaron, M.D., F.R.C.S. (Edin.), F.R.C.S. (Eng.) Assistant Surgeon, Royal Infirmary, Edinburgh, Scotland, Cloth. Price $7. Ed. 3. Pp. 390, with 61 illustrations. Baltimore, 1949. Williams & Wilkins Company.

COMPANION IN SURGICAL STUDIES. By Ian Aird, Ch.M., F.R.C.S. Professor of Surgery, University of London. Cloth. Price $15. Pp. 1060. Baltimore, 1949, William & Wilkins Company.

TRANSACTIONS OF THE THIRD AMERICAN CONGRESS ON OBSTETRICS AND GYNECOLOGY. By George W. Komak, M.D., New York, and Robert N. Rutherford, M.D. Cloth. Price $9. Pp. 412, with 64 illustrations. Portland, Ore., 1948, Bernciiff Printers.

PHYSICS AND THE SURGEON. By H. S. Souttar, D.M., Ch. (Oxon), F.R.C.S. (Eng.), Consulting Surgeon, London Hospital. Leather. Pp. 60, with 41 illustrations. Springfield, Illinois, 1948, Charles C Thomas, Publisher.

EARLY CARCINOMA OF THE UTERINE CERVIX. By Hansjakob Wespi, M.D., Chief of the Obstetrical and Gynecological Department, Frauenfeld Hospital, Aarau, Switzerland. Cloth. Price $6.50. Pp. 271, with 95 illustrations. New York, 1949, Grune & Stratton, Inc.

TOM CULLEN OF BALTIMORE. By Judith Robinson, Toronto, Canada. Cloth. Price $3.50. Pp. 435, with 1 illustration. Toronto, 1949, Oxford University Press.

TEXTBOOK OF SURGERY. By Frederick Christopher, B.S., M.D., F.A.C.S., Professor of Surgery, Northwestern University Medical School, Evanston, Ill. Cloth. Ed. 5. Pp. 1550, with 1465 illustrations. Philadelphia, 1949, W. B. Saunders Company.

UROLOGICAL ASPECTS OF SPINAL CORD INJURIES. By George C. Prather, M.D., Department of Genito-Urinary Surgery, Harvard Medical School, Boston. Leather, Price $3.75. Pp. 143, with 42 illustrations. Springfield, Ill, 1949. Charles C Thomas, Publisher.

OPERATIVE TECHNIC. Edited by Warren H. Cole, Department of Surgery, University of Illinois, Urbana, Ill. Cloth. Pp. 725, with 394 illustrations. New York, 1949, Appleton-Century-Crofts Company, Inc.

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AN ATLAS OF THE BLOOD AND BONE MARROW. By R. Phillip Custer, M.D., Director, Laboratories of the Presbyterian Hospital in Philadelphia; Assistant Professor of Pathology, the University of Pennsylvania School of Medicine; Consultant to the Armed Forces Institute of Pathology. Cloth. Pp. 321, with 285 illustrations. Philadelphia, 1949, W. B. Saunders Company.

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SKIN GRAFTING. By James Barrett Brown, M.D., Professor of Clinical Surgery, Washington University School of Medicine, and Frank McDowell, M.D., Assistant Professor of Clinical Surgery, Washington University School of Medicine. Cloth, Ed. 2. Pp. 340, with 239 illustrations. Philadelphia, 1949, J. B. Lippincott Company.

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BRITISH SURGICAL PRACTICE. Vol. V. By Sir Ernest Rock Carling, F.R.C.S., F.R.C.P., Consulting Surgeon Westminster Hospital, and Sir J. Paterson Ross, M.S., F.R.C.S., Surgeon and Director of Surgical Clinical Unit, St. Bartholomews Hospital; Professor of Surgery, University of London. Paper. Pp. 494, with many illustrations. London, England, 1948, Butterworth & Co., Ltd. (St. Louis, The C. V. Mosby Company).

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SURGICAL TREATMENT FOR ABNORMALITIES OF THE HEART AND GREAT VESSELS. By Robert E. Gross and William E. Ladd, Professor of Child Surgery, Harvard University Medical School, Boston. Soft. Price $2. Pp. 72, with several illustrations. Springfield, Ill., 1950, Charles C Thomas, Publisher.

MENSTRUATION AND ITS DISORDERS, PROCEEDINGS OF THE CONFERENCE HELD UNDER THE AUSPICES OF THE NATIONAL COMMITTEE ON MATERNAL HEALTH. Edited by Earl T. Engle. Hard. Pp. 338, with illustrations. Price $6.50. Springfield, Ill., 1950, Charles C Thomas, Publisher.

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ANNOUNCEMENTS

The Kappa Delta Award for Research in Orthopaedic Surgery

A prize of $1,000, donated by the Kappa Delta Sorority, is awarded annually by the American Academy of Orthopaedic Surgeons for the best research done in orthopaedic surgery or a related scientific field by an individual in the United States. The second award, for the year 1950, will be presented at the eighteenth annual convention of the Academy in Chicago, Jan. 31, 1951. Researchers interested in competing for this prize are requested to secure further information from Dr. R. Beverly Raney, Duke Hospital, Durham. N. C., Chairman of the Award Committee for 1950.

Third Annual Symposium on Orthopedic Appliances

According to an announcement just made by Dr. E. R. Weidlein, Director of Mellon Institute, plans are well under way to convene in Pittsburgh for the Third Annual Symposium on Orthopedic Appliances. The meetings have been set for the week of Sept. 18, 1950. This symposium will once more be a cooperative undertaking of the Department of Orthopedic Surgery of the University of Pittsburgh's School of Medicine and Mellon Institute's Multiple Fellowship on Orthopedic Appliances sponsored by the Sarah Mellon Scaife Foundation.

In contrast to the programs of the first and second annual symposiums, the 1950 sessions will probably be devoted almost exclusively to laboratory-, shop-, and clinic-type demonstrations and discussions, with emphasis on active participation by all those in attendance.

Inquiries concerning final program details and requirements for attendance should be addressed to the Orthopedic Appliances Fellowship, Mellon Institute, 4400 Fifth Avenue, Pittsburgh 13, Pa.

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